VOLUME 57 JANUARY, 1947 NUMBER 1

MEDICAL LIBRARY FEB 12 1947

[FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY]

The AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

OFFICIAL ORGAN
THE AMERICAN ROENTGEN RAY SOCIETY
THE AMERICAN RADIUM SOCIETY

Editor: LAWRENCE REYNOLDS, M.D.

Editorial Board

A. C. CHRISTIE, M.D. E. H. SKINNER, M.D. LAURISTON S. TAYLOR

Advisory Board for Pathology

EUGENE L. OPIE, M.D.

ISSUED MONTHLY FOR THE AMERICAN ROENTGEN RAY SOCIETY
BY CHARLES C THOMAS, PUBLISHER, SPRINGFIELD, ILLINOIS

American Roentgen Ray Society • *Forty-eighth Annual Meeting*
Haddon Hall, Atlantic City, New Jersey
September 16-19, 1947

American Radium Society • *Twenty-ninth Annual Meeting*
Seaside Hotel, Atlantic City, New Jersey
June 9-10, 1947

YEARLY SUBSCRIPTION, $10.00 S

DUPONT
X-RAY
SAFETY
FILM
Xtra-fast
This film has been made with great care, and though it will be promptly replaced if found defective in manufacture, it is sold without express or implied warranty or other liability of any kind.
E. I. DU PONT DE NEMOURS & COMPANY, (INC.)
PHOTO PRODUCTS DEPARTMENT
Factory: Parlin, New Jersey
TYPE 508

FEATURES

- Clear blue safety base
- ntrast
- latitude
- Convenient packaging
- Speed
- Detail

E. I. du Pont de Nemours & Co. (Inc.)
Photo Products Department
Wilmington 98, Delaware
In Canada: Canadian Industries, Ltd.

PONT X-RAY FILM and CHEMICALS

TER THINGS FOR BETTER LIVING . . . *THROUGH CHEMISTRY*

DUPONT
REG. U. S. PAT. OFF.

ANDREW CONWAY IVY

Caldwell Lecturer, 1946

UNIV. OF MICH.

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 57 JANUARY, 1947 NO. 1

MOTOR DYSFUNCTION OF THE BILIARY TRACT

AN ANALYTICAL AND CRITICAL CONSIDERATION

CALDWELL LECTURE 1946*

By A. C. IVY, PH.D., M.D.

Vice-President, University of Illinois in charge of the Chicago Professional Schools and Distinguished Professor of Physiology in the Graduate School

CHICAGO, ILLINOIS

INTRODUCTION

MR. PRESIDENT, and Members of The American Roentgen Ray Society: It is a signal and high privilege to be invited to meet with you to recall and honor the name and works of one of the martyrs of medicine and outstanding contributors to the science of roentgenology.

We shall honor him best by rededicating ourselves to emulate the conscientiousness and skill with which he performed his daily tasks and the enthusiasm with which he explored "pure" science for the purpose of improving roentgenological apparatus and methods of diagnosis. If medical science is to progress, these attitudes must prevail on a par with those engendered by the need of earning our daily bread and shelter.

Accordingly, in this lecture it is my aim to present to you an important clinical diagnostic question which requires an answer. Its answer will require the ingenuity, perseverance and critical ability of a Dr. Eugene W. Caldwell and the cooperation of the roentgenologist, internist and surgeon.

The question is: Does biliary dyskinesia (ill motion) exist as a clinical entity; and, if so, how can it be accurately diagnosed? The question may be stated also as follows: Do motor dysfunctions of the extrahepatic biliary tract occur to the extent that symptoms result? The answer to this latter question clearly implies that we must be able to diagnose a motor dysfunction of the extrahepatic biliary tract. The answer to this question is requisite to achieve the implications of the title of a paper published by Dr. Caldwell[14] in 1915. The title of the paper is "Safe Interpretations of Roentgenograms of the Gall Bladder Region."

I shall first outline the anatomy and physiology of the extrahepatic biliary tract and then critically analyze the available evidence which suggests that motor dysfunction of the biliary tract may exist.

* Delivered before the Forty-seventh Annual Meeting of the American Roentgen Ray Society, Cincinnati, Ohio, Sept. 17–20, 1946.

ANATOMY

Only those structures which directly pertain to the motor activity of the biliary tract will be mentioned.

The gallbladder is known to have a fibromuscular layer. A sphincter has been described at the junction of the gallbladder with the cystic duct in some individuals,[19,34] though this has not been confirmed by others.[27,44] Muscular fibers are usually very sparse in the cystic, hepatic, and common bile duct of man.

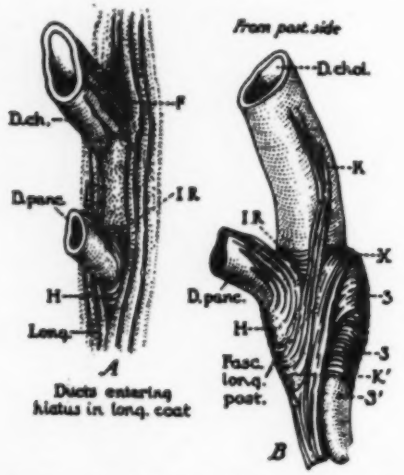


FIG. 1. Sketches of Hendrickson's maceration specimens, redrawn from his article in the *Bull. Johns Hopkins Hosp.*, Vol. 9 (ref. 53).

Our knowledge of the anatomy of the intramural portion of the common duct has been extended in recent years by Boyden and his colleagues. The embryological evidence shows that the various portions of the sphincter of Oddi develop as a true musculus proprius of the ducts independent of the musculature of the duodenum.[53] More recently Boyden has summarized the results of their studies on the fetal and adult choledochoduodenal junction in man.[29] In the adult there are four intrinsic muscles of the ducts and ampulla (Fig. 1). *First*, there are two longitudinal bundles (the longitudinal fascicles) which tend to shorten the duct and erect the papilla. *Second*, there is an annular sheath of muscle which surrounds the common bile duct from the point where it enters the duodenal wall to where it joins the pancreatic duct (the sphincter choledochus). *Third*, an annular sheath which surrounds the terminal portion of the pancreatic duct before it joins the common duct (the sphincter pancreaticus) and is present in about one-third of adults. *Fourth*, an annular sheath which surrounds the ampulla and is well defined in about one-sixth of adults.[29]

It must be kept in mind that the anatomical relation of the common bile duct to the pancreatic duct is subject to considerable variation. The two ducts open separately in 29 per cent of cases in man (Table 1).[1,2,29,45,47,50] When the two ducts join, the length of the common ampulla is 3 mm. or more in only 20 per cent of individuals.[38] This figure of 20 per cent is significant, since it represents the percentage of persons in whom reflux of bile and pancreatic juice into the respective ducts may occur. Negri[42] interprets his observations as indicating that reflux is possible in 88 per cent of persons; i.e. he finds that the two ducts join before the papilla in 88 per cent of individuals. Actually pancreatobiliary reflux has been found to occur during cholangiography in only 20 per cent of 91 cases.[31]

TABLE I

FREQUENCY OF SEPARATION OF THE ORIFICE OF THE COMMON BILE AND PANCREATIC DUCTS IN MAN

Author	Number of Cases Examined	Separate Opening Found	
		No. of Cases	Per Cent
Opie	100	11	11
Baldwin	90	20	22
Schirmer	48	22	47
Belou	50	27	54
Mann and Giordano	200	62	31
Rienhoff and Pickrell	250	73	29
TOTAL	738	215	29

Reflux of opaque fluid into the pancreatic duct occurred during cholangiography in 21 per cent of 91 cases.[31]

Thus, anatomically the basis of a motor mechanism exists in the gallbladder and in the intramural portion of the common bile duct. And, any motor mechanism may become disordered.

Pathological Anatomy. Nothing of significance can be said regarding the pathological anatomy of the musculature of the upper biliary tract. On the contrary, hypertrophy of the sphincter of Oddi has been reported by a number of investigators[3,43,44,58] and two specimens have been published by Mann and Giordano.[38] Hypertrophy of the sphincter has been reported to occur after cholecystectomy,[44,58] though this certainly has not been proved.

PHYSIOLOGY

The Gallbladder. There is no sound reason to doubt today that the gallbladder evacuates by way of the cystic duct under the force of its contraction which is caused primarily by the action of the hormone cholecystokinin. The supporting evidence has been reviewed by me[22] and more recently by Hill.[21] Though the motor activity of the gallbladder of some species is influenced by the vagus and splanchnic nerves, there is no good evidence that such is true of the gallbladder of man and the dog.

The hormone cholecystokinin is produced by the upper intestinal mucosa when in contact with acid, fat or partially digested protein. It is not produced in response to a carbohydrate meal or water, as judged from the lack of effect of these substances on the evacuation of the gallbladder.[17,21,55] When injected intravenously it causes evacuation of the gallbladder in human and canine subjects.[23] It has only been used in 8 human subjects to date in conjunction with cholecystography because we have not produced a preparation of the hormone which is entirely safe when given intravenously.

The maximum force which the contracting gallbladder exerts on its contents has not been measured in man. In the dog it amounts to from 20 to 25 cm. of water or bile pressure. This is somewhat less than the secretory pressure of bile and pancreatic juice which ranges from 25 to 30 cm. of water pressure. These figures are important to bear in mind in considering the elicitation of symptoms by the distention of the biliary tract.

The Sphincter of Oddi. Only those points regarding the physiology of the sphincter of Oddi which are most pertinent to the present discussion will be referred to here.

Anatomically a sphincter independent of the duodenal musculature appears to exist. The apparently conflicting physiological observations warrant the following conclusion: In all animals that have a gallbladder the common duct possesses a sphincteric mechanism, which is intimately coordinated with duodenal tone and motility but which may also act independently. The most recent evidence on this point has been discussed by Bergh and Layne[5] and Long.[32] These authors present convincing evidence that the choledochal sphincter can act independently of the duodenal wall in man and the dog.

The sphincteric mechanism can exert a resistance to the flow of fluid into the duodenum amounting to 60 cm. of water pressure in man and to 80 cm. of water pressure in the dog. It is possible, then, for a spastic sphincter to exert a resistance to flow greater than the capacity of the gallbladder to contract and greater than the secretory pressure of the liver. Thus, if the gallbladder rapidly contracted against a spastic sphincter, pain might result.

The normal resistance of the sphincter in the cholecystectomized human subject ranges from 0 to 23 cm. of water pressure; it usually ranges from 10 to 15 cm. of water.[18,30,36]

In the dog stimulation of the vagus nerve increases the resistance to the flow of bile into the duodenum.[33] Because of the complexity of the problem, it is unwise to draw an unqualified conclusion regarding the functional innervation of the sphincter. In the dog, Goldman and Ivy[20,24] produced a reflex contraction of the sphincter by stimulating the colonic nerve or by sud-

denly distending the colon to simulate the distress of a "spastic" colon. To my knowledge, no attempt has been made to produce in man a reflex hypertonus or spasm of the sphincter of Oddi by distending the colon to the point that distress is produced.

In the dog the reciprocal innervation of the gallbladder and sphincter has been demonstrated to the extent that after a ation of the gallbladder or give rise to increases in intrabiliary tract pressure sufficient to cause symptoms.

Bergh[4] reports that "in patients with inflammatory lesions, such as cholangeitis secondary to stone, it is not uncommon for the sphincter to remain irritable for weeks after removal of the stone by operative procedure. In such cases the sphincter

TABLE II

Sample	Time min.	Volume cc.	Flow per minute cc.	Lipase Total per min. units	Bilirubin mg. per 100 cc.	Cholesterol mg. per 100 cc.	Remarks
Control							
2	12	15	1.25	138	0.8	Trace	
3	23	5	0.22	54	None	None	Bile flow ceased
Injection of Secretin-Cholecystokinin (Dog doses 300 S and 10 C)							
4	5	20.5	4.1	869	None	None	
5	3	15.5	5.2	768	None	None	
6	3	14.0	4.7	733	None	None	
7	2	8.0	4.0	584	None	None	Rt. hypoch. distress
8	5	18.0	3.6	331	Trace*	Trace*	Rt. hypoch. distress
9	13	37.0	2.9	220	None	None	Rt. hypoch. distress
10	4	9.5	2.4	235	None	None	Ache under scapula
11	7	19.0	2.7	454	None	None	Pain increasing
12	7	21.0	3.0	294	None	None	About the same
Totals	49 min.	162.5					
50 cc. 33% $MgSO_4$ injected into duodenum. Period of injection about 4 min.							
13	4	18.5	4.6	539	42.0	44.3	3.58 $MgSO_4$ % in drainage Pain disappeared
14	3	18.5	6.2	830	92.8	83.3	0.50 $MgSO_4$ % in drainage
15	3	13.0	4.3	326	88.2	79.6	0.66 $MgSO_4$ % in drainage
16	4	7.0	1.8	265	27.0	27.0	0.44 $MgSO_4$ % in drainage

* Too small to determine quantitatively.

meal when the gallbladder contracts the sphincter relaxes.[21,22] Also, cholecystokinin apparently causes relaxation of the sphincter.[49] In cholecystectomized patients with a T-tube in the common duct, Bergh[4] observed relaxation of the sphincter after a fatty meal in 13 cases and no change after a carbohydrate meal in 8 cases. Doubilet and Colp[18] obtained variable results after a mixed meal. Theoretically a disturbance of reciprocal innervation could delay evacu- readily goes into spasm and the intramural resistance may be thirty, forty or fifty centimeters of water or even higher."

There are two drugs which quite uniformly cause a spasm of the sphincter or an increased resistance to the flow of bile into the duodenum in man and the dog. These are morphine and pilocarpine.[4,13,18,33] In patients with a T-tube in place magnesium sulphate introduced into the duodenum causes relaxation of the sphincter.[4,18] The

same is generally true of such drugs as amyl nitrate, nitroglycerine, and theophylline ethylenediamine.[4,7,8,13] Atropine produces relaxation in the dog; it seems to do so occasionally in man,[9,12,46] but usually has little effect on spasm of the sphincter, especially that induced by morphine.[4,13]

THE SOURCE OF PAIN FROM THE NON-INFLAMED BILIARY PASSAGES

It is evident and well established by the foregoing evidence that the sphincter of Oddi or the choledochoduodenal mechanism can contract with sufficient force to prevent evacuation of the contracting gallbladder and to counteract the secretory pressure of the liver. In addition, on the basis of the anatomic and physiologic mechanisms concerned in the normal evacuation of the extrahepatic biliary passages, it is possible for abnormalities to occur.

As in the case of other hollow viscera when motor dysfunction occurs, distress may arise from abnormal distention or from localized spasm. So the question arises: *Can distress arise from the degree of distention possible in the non-inflamed passages?*

Evidence from Experiments on the Recently Operated Animal or Patient. In the dog Schrager and Ivy[52] found that the minimal pressure required to elicit pain on distention of the bile ducts was 27 cm. of water. That is, the pressure required to elicit pain by distention of the bile ducts is approximately equivalent to the maximal force of the gallbladder contraction and the secretory pressure of bile. In human subjects with a T-tube in place a rather sudden increase in pressure in the duct, ranging from 15 to 50 cm. of water, will cause pain[30,36,37] typical for biliary colic. Gradual distention up to 100 cm. of water does not cause pain.[30] Among the cases studied by Walters and his colleagues[36,37] I have been able to find only 2 cases in which pain was produced by pressures within the range of physiological values. In one of these, pain was first produced by a pressure of 15 cm. of water and then two months later 50 cm. of water pressure was required. This indicates that the threshold of the pain nerves was lowered by the presence of an inflammatory process which later disappeared. In the studies of Layne and Bergh[5,30] on 30 patients the pressure was suddenly elevated to 50 cm. of water. This caused typical biliary tract pain in every subject but one. They made no attempt, however, to determine the pressure-pain threshold. Thus, in the case of man the evidence showing that pain may result from distention of the bile duct with physiologically possible pressures is relatively meager. When pain has occurred the ducts have been in such condition that it could be said that the pain threshold was lowered by inflammation.

Can pain arise from spasm of the sphincter of Oddi alone? It seems to have been clearly demonstrated in cholecystectomized patients with a T-tube in the common bile duct that spasm of the sphincter alone can cause pain varying from a mild ache to severe and intermittent colicky pain. This is clear from the observations of Walters and his colleagues,[13,35,36,54,56] Best and Hicken,[7] and Layne and Bergh.[5,30] For example, Bergh and Layne[5,30] could produce a spasm of the sphincter and pain by sudden distention of the duct with a pressure of 50 cm. of water. The pressure could then be withdrawn, but the pain and evidence of spasm, as indicated by increased intramural resistance, would continue for several minutes. Their kymographic records constitute impressive evidence of the correlation between spasm of the sphincter and intermittent changes in intensity of the pain. In addition, Walters and his colleagues[13,35,36,54,56] found that in patients with the syndrome of postcholecystectomy colic, the administration of ⅛ grain or less morphine would cause an attack of biliary colic. This attack of colic was associated with an increase in intramural resistance up to 50 to 80 cm. of water pressure. Walters and Snell write: "This phenomenon is attributed to a hyperkinetic sphincter of Oddi which is thrown into a state of tonic

contraction by morphine." I know of 5 patients, who have been cholecystectomized for biliary colic, in whom morphine will induce an attack of colic. Walters and Snell[56] found some cases in which the apparent spasm of the sphincter was associated with a local spasm of the duodenum. However, Bergh and Layne[5] studied 5 cases, likewise with a balloon in the duodenum and a T-tube in the common duct, and obtained pain with spasm of the sphincter without a contraction of the duodenum.

I can offer no other explanation for the evidence obtained by these investigators than that a spasm of the sphincter *per se* can give rise to pain. Since this is the case and since rapid distention may cause spasm of the sphincter, it cannot be concluded to what extent distention of the ducts *per se* contributes to pain. Nevertheless it is clear that in a few cases the distention of the duct with pressures in the physiological range has caused pain.

Evidence from Experiments on Normal or Non-recently Operated Patients. There are three different experiments which show that pain may occur from procedures that presumably cause distention of the ducts by physiological pressures in the absence of a T-tube or other foreign body. These experiments are crucial since they apply to the non-recently operated biliary tract. The *first experiment* was performed by Westphal.[59] He passed a duodenal tube and initiated a contraction of the gallbladder by introducing olive oil. He then injected pilocarpine, a drug which causes the sphincter to contract, and noted that the flow of bile into the duodenum ceased and biliary tract distress occurred. An injection of atropine relieved the distress which was associated with a return of the flow of bile into the duodenum. The only criticism of this experiment is that the distress might be attributed to spasm of some other portion of the alimentary tract. The *second experiment* was reported by von Bergmann.[6] He observed that the intravenous injection of dehydrocholic acid, which causes a brisk formation of bile by the liver, into patients with a stenosis of the common duct causes pain. This would appear to be crucial evidence, but it requires confirmation. Oldberg and I[25] made a somewhat similar observation on the dog. When we injected cholecystokinin into a dog with the cystic duct obstructed, pain-distress with nausea and vomiting occurred. The *third experiment* first occurred as an accident when Ivy, Voegtlin and Greengard[26] were injecting intravenously a preparation containing secretin and cholecystokinin. The experiment may be outlined as follows: A duodenal tube was passed and a flow of bile-stained fluid was obtained. Then the bile disappeared from the drainage fluid which was obviously due to closure of the sphincter choledochus. A dose of secretin-cholecystokinin was injected and a copious flow of pancreatic juice unstained with bile occurred. About ten minutes later, right hypochondrial distress was experienced. The distress increased and spread to the right scapula. Then 50 cc. of 33 per cent magnesium sulphate was injected into the duodenum. The pain disappeared in four minutes and dark bile-stained fluid was obtained from the duodenum. This experience occurred in 3 of 19 subjects. The data from one experiment are shown in Table 1.

Other Sites in the Biliary Passage Alleged to Manifest Spasm. It has been claimed that a sphincter exists at the junction of the neck of the gallbladder with the cystic duct (collum cysticus sphincter).[22,34] It has been claimed that spasm or hypertonus of this sphincter causes annular bands of contraction to appear in the gallbladder without evacuation in response to a fatty meal. This is in contrast to a spasm of the spincter of Oddi in which the gallbladder evacuates to some extent and the hepatic ducts are visualized.[12,28,48,51] Recently Mirizzi[39,40] described a sphincter of the common hepatic duct and presented cholangiograms purporting to show cases of spasm of the sphincter. However, from the evidence presented one cannot be certain that the constriction was due to spasm or organic stricture.

VISUALIZATION OF THE COMMON BILE DUCT AND PANCREATIC DUCT: PANCREATOBILIARY REFLUX

Numerous articles have appeared in which the pancreatic and common bile duct have been visualized during cholangiography. For example, the opaque oil may start to flow into the duodenum, then the flow stops and both ducts become visualized. Then after a variable period, the ducts empty. This reflux was observed to occur in 21 per cent of 91 patients.[31] Though this reflux is attributed to spasm of the distal portion of the ampulla (Fig. 2), the presence of pain is not mentioned or emphasized by any of the authors reporting on this subject. So visualization of the pancreatic duct or spasm of the distal portion of the sphincter of the ampulla does not characteristically cause pain.

Some believe that the reflux into the pancreatic duct is of no significance;[39,40] others believe that it is associated with symptoms and causes anatomical changes.[41]

DIAGNOSIS

Though plenty of evidence is available to prove that motor dysfunction of the sphincter of Oddi may occur and cause symptoms, there is no certain way to diagnose the condition at the present time in the absence of manometric studies. Hill[21] in his discussion of the problem writes: "The hypertonic and atonic types of dyskinesia or dyssynergia may be detected only by a correlation of a carefully taken history with the findings obtained from duodenal intubation and roentgenological studies." Walters and Snell[56] grant that there is a good basis for syndromes of motor dysfunction but state that "further clinical experience may serve to establish them as definite entities, but for the present the matter must be held sub judice."

There are really two problems in the diagnosis of motor dysfunction of the passages. These are the diagnosis in the presence of the gallbladder and in the cholecystectomized patient. We shall consider the former problem first.

The Diagnosis in the Presence of a Gallbladder which Concentrates.

Delayed Evacuation. Delayed evacuation of the gallbladder after a fatty meal has been said to be characteristic of the hypertonic type of dyskinesia by Newman[43] and Westphal.[60] It is reasonable to suppose, however, that a hyperkinetic or excessively active gallbladder contracting against only the slight resistance of the sphincter could cause pain.

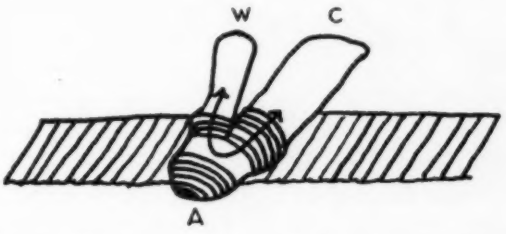


FIG. 2. Diagram of sphincteric arrangement which would permit pancreatobiliary reflux. Such reflux occurred in 21 per cent of 91 cases during cholangiography.[31]

The difficulty in using evacuation time as a criterion of the normality of the motor activity of the biliary passages is the variability observed and the relatively meager work that has been done on the subject. Case[15] studied 36 normal subjects and found that the shadow was reduced at least 90 per cent within three hours after a fatty meal. In Table III I have collected Boyden's data on 76 presumably normal subjects in whom roentgenograms were made at frequent intervals after a fatty meal. It is to be noted that forty minutes after the fatty meal the shadow was reduced 75 per cent on the average but the range was from 28 to 100 per cent.

Whether the variability observed is due to a difference in the motor activity of the gallbladder or to a difference in the rate of evacuation of the fat from the stomach is unknown. This, however, is an important question if we are ever to satisfactorily use evacuation time as a criterion of the existence of a functional motor disturbance. The only solution which has occurred to me is to give a standard stimulus paren-

terally. Cholecystokinin would be such a stimulus.

Until cholecystokinin becomes available, it would be worth while to correlate the rate of evacuation of the gallbladder with the history of the biliary tract type of distress.

TABLE III

RATE OF EVACUATION OF THE GALLBLADDER IN NORMAL SUBJECTS AFTER 4 EGG YOLKS IN THE ADULTS AND 3 IN THE CHILDREN

No. of Subjects	Sex	Age Year	Per Cent Evacuation at 20 min. Average	20 min. Range	40 min. Average	40 min. Range
10	F	60–78	73	55–87	84	64–97
14	M	60–63	49	21–84	73	50–94
12	F	17–34	58	17–93	73	28–93
12	M	18–28	43	3–72	64	43–86
12	F	6.5–11.5	55	11–67	77	57–100
16	M	6.0–11.3	70	39–88	82	70–97
76	All ages and sex		58	3–93	75	28–100

In Boyden's series of presumably normal subjects which visualized, only 2 of 84 failed to empty some in 45 minutes. (Boyden, E. A., and Fuller, A. H. *Am. J. Dis. Child.*, 1934, *48*, 565; Boyden, E. A., and Grantham, S. A. *Surg., Gynec. & Obst.*, 1936, *62*, 34.)

The results of a good study of this type are not available in the literature.

Visualization of the Bile Ducts. Sussman[55] reports that if three egg yolks in some milk are given in the presence of good visualization of the gallbladder, *the cystic and common duct* can be visualized in 75 per cent of cases in which emptying occurred. He made roentgenograms at ten, twenty and thirty minutes. This is what should be expected if the gallbladder contracts well and squeezes out concentrated, roentgen opaque bile at a rate so that dilution in the ducts by hepatic bile does not occur.

Visualization of the hepatic ducts is unusual. Bronner[12] observed it in none of 49 normal subjects and in 4 cases of peptic ulcer. He records that only one patient had pain when the hepatic ducts visualized and that the pain disappeared after treatment with atropine. I have never seen the hepatic ducts of the dog become visualized when a fatty meal or cholecystokinin was given to cause gallbladder evacuation. In man I have seen 6 cases of visualization of the hepatic ducts, through the courtesy of Doctors E. E. Barth and J. T. Case (Department of Radiology, Northwestern University), but only one had distress at the time. Copleman and Sussman[16] have reported 4 cases; however, they do not state that distress occurred at the time of the visualization, though the patients were subjected to cholecystography because they had biliary tract symptoms.

It seems certain that visualization of the hepatic ducts means that the gallbladder is expelling concentrated bile faster than the bile could enter the intestine. If this is associated with distress and pain at the time, the circumstantial evidence for dyskinesia is strong. If it occurs in the absence of pain, then the pressure in the ducts or the spasm of the sphincter is inadequate to cause pain.

This reasoning appeared to be clear until Boyden and Layne[10] reported that in 7 of 26 patients with pernicious anemia the hepatic ducts became visualized in from eight to sixteen minutes after the ingestion of egg yolks. Dr. Boyden has informed me that none of the 7 patients had any distress and for that reason they believed the visualization of the hepatic ducts was due to a "permanent narrowing of the common duct." This would indicate that they believe the pain in dyskinesia arises from spasm of the sphincter and not from distention of the ducts by the contracting gallbladder.

Thus, visualization of the hepatic ducts with a normal gallbladder *in situ* may occur with or without pain, generally the latter. However, we are not certain regarding the explanation of this observation.

Suggestions. It is believed on the basis of our experience with 3 of 19 subjects that the

use of cholecystokinin should expose for diagnosis latent cases of dyskinesia. This procedure associated with cholecystography (and perhaps visualization of the hepatic ducts) may supply the crucial clinical and roentgenological evidence for an accurate diagnosis.

Another suggestion regarding the problem arises from the report of Sussman that morphine markedly retards the evacuation of the gallbladder. This should be expected in view of the effect of morphine on the sphincter of Oddi, but the drug may act by delaying the gastric evacuation of the meal of egg yolks. This could be avoided by introducing the egg yolk into the duodenum by tube and then the susceptibility of the sphincter of Oddi of the patient to spasm induced by morphine could be determined. Such a method is not very practical, however.

It would be well to know in how many cases in which the internist finds typical biliary colic, the roentgenologist reports a normal cholecystogram. Information of this kind would serve to indicate how frequently pain due to motor dysfunction may occur.

Post-cholecystectomy Colic. The diagnosis of spasm of the sphincter prior to operation is more difficult in the cholecystectomized than in the non-cholecystectomized patient. Weir and Snell,[57] after listing the organic causes of post-cholecystectomy colic, write that in the presence of unexplained pain resembling biliary colic and not referable to any gross disease of the liver or bile ducts, it is necessary to assume the presence of spasm of the sphincter and transient physiological obstruction.

In some cases the induction of colic with a sixth of a grain of morphine may prove useful, since I know of no other condition where morphine induces a colic, though it may at times increase ureteral colic after the colic has started.

A dye, more roentgen opaque than those we use at present, which would be excreted by the liver in sufficient quantities to visualize the ducts without concentration, should help considerably in the diagnosis of post-cholecystectomy colic.

SUMMARY

It has been established that the sphincter choledochus or choledochoduodenal mechanism can contract in man with sufficient force to prevent the evacuation of the contracting gallbladder and to counteract the secretory pressure of bile.

Convincing evidence is available showing that pain may arise (*a*) from the rather sudden distention of the biliary passages by a force within the range of the contractile power of the gallbladder and the secretory pressure of bile, and (*b*) from a spasm of the sphincter choledochus and/or the adjacent duodenum.

There is adequate evidence of a physiological nature, provided by a large group of investigators, to justify the view that a motor dysfunction of the biliary passages may produce symptoms, or that such an entity as biliary dyskinesia exists.

In the absence of manometric studies via a choledochostomy the clinical diagnosis of biliary diskinesia is difficult, if not impossible, to prove. The strongest circumstantial evidence in support of the diagnosis in the presence of a gallbladder which visualizes is the visualization of the hepatic ducts with pain after the ingestion of a fatty meal. This combination has only rarely been observed.

The diagnosis of biliary dyskinesia in the patient with or without a gallbladder is a challenging roentgenological and clinical problem. There is reason to hope that the isolation and use of cholecystokinin will assist in the solution of the problem.

1853 West Polk St.
Chicago 12, Illinois

REFERENCES

1. Baldwin, W. M. *Anat. Rec.*, 1911, *5*, 197.
2. Belóu, P. Cited by Mann and Giordano.
3. Berg, J. *Acta chir. Scandinav.*, Suppl. 2, 1922, p. 1.
4. Bergh, G. S. *Staff Meet. Bull., Univ. of Minnesota Hosp.*, 1938, *10*, 73.

5. BERGH, G. S., and LAYNE, J. A. Demonstration of independent contraction of spincter of common bile duct in human subjects. *Am. J. Physiol.*, 1940, *128*, 690–694.
6. VON BERGMANN, G., and GOLDNER, M. Funktionelle Pathologie. J. Springer, Berlin, 1932, Chapt. 5.
7. BEST, R. R., and HICKEN, N. F. Biliary dyssynergia; physiological obstruction of common bile duct. *Surg., Gynec. & Obst.*, 1935, *61*, 721–734.
8. BEST, R. R., and HICKEN, N. F. Biliary dyssynergia; cholangiographic recognition and its significance. *West. J. Surg.*, 1936, *44*, 467–473.
9. BEST, R. R., and HICKEN, N. F. Cholangiographic demonstration of biliary dyssynergia and other obstructive lesions of gallbladder and bile ducts. *J.A.M.A.*, 1936, *107*, 1615–1620.
10. BOYDEN, E. A., and LAYNE, J. A. Gall bladder in patients with pernicious anemia; study of nonvisualization and rate of emptying. *Gastroenterology*, 1945, *4*, 121–134.
11. BOYDEN, E. A., and RIGLER, L. G. Cholecystographic and fluoroscopic study of reaction of human gall bladder to faradic stimulation of stomach and duodenum. *Anat. Rec.*, 1934, *59*, 427–447.
12. BRONNER, H. Die cholezystographische Motilitätsprüfung der Gallenblase und ihre Ergebnisse. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1929, *39*, 23–76.
13. BUTSCH, W. L., McGOWAN, J. M., and WALTERS, W. Clinical studies on influence of certain drugs in relation to biliary pain and to variations in intrabiliary pressure. *Surg., Gynec. & Obst.*, 1936, *63*, 451–456.
14. CALDWELL, E. W. Safe interpretation of roentgenograms of the gall bladder region. AM. J. ROENTGENOL., 1914–1915, *2*, 816–819.
15. CASE, J. T. Evaluation of cholecystography. *J.A.M.A.*, 1929, *92*, 291–297.
16. COPLEMAN, B., and SUSSMAN, M. L. Hepatic duct visualization following oral cholecystography. *Radiology*, 1941, *36*, 465–467.
17. CRANDALL, L. A., JR. Mechanisms of contraction and evacuation of gallbladder. *Arch. Int. Med.*, 1931, *48*, 1217–1224.
18. DOUBILET, H., and COLP, R. Resistance of sphincter of Oddi in human. *Surg., Gynec. & Obst.*, 1937, *64*, 622–633.
19. GLISSON, F. Anatomia Hepatis. London, 1681. Cited by Boyden, E. A. *Anat. Rec.*, 1936, *66*, 217.
20. GOLDMAN, L., and IVY, A. C. Effect of distention of colon and stimulation of its nerve supply on flow of bile from liver. *Ann. Surg.*, 1939, *110*, 755–765.
21. HILL, H. A. Functional disease of extra-hepatic biliary system. *Radiology*, 1937, *29*, 261–278.
22. IVY, A. C. Physiology of the gallbladder. *Physiol. Rev.*, 1934, *14*, 1–102.
23. IVY, A. C., DREWYER, G. E., and ORNDOFF, B. H. Effect of cholecystokinin on human gall bladder. *Endocrinology*, 1930, *14*, 343–348.
24. IVY, A. C., and GOLDMAN, L. Physiology of biliary tract. *J.A.M.A.*, 1939, *113*, 2413–2417.
25. IVY, A. C., and OLDBERG, E. Hormone mechanism for gall-bladder contraction and evacuation. *Am. J. Physiol.*, 1928, *86*, 599–613.
26. IVY, A. C., VOEGTLIN, W. L., and GREENGARD, H. Physiology of common bile duct. *J.A.M.A.*, 1933, *100*, 1319–1320.
27. JOHNSTON, C. G., and BROWN, C. E. Studies of gallbladder function. *Surg., Gynec. & Obst.*, 1932, *54*, 477–485.
28. KALK, H., and SCHÖNDUBE, W. Ueber die Funktion der Gallenblase. *Ztschr. f. d. ges. exper. Med.*, 1926, *53*, 461–483.
29. KREILKAMP, B. L., and BOYDEN, E. A. Variability in composition of sphincter of Oddi. *Anat. Rec.*, 1940, *76*, 485–497.
30. LAYNE, J. A., and BERGH, G. S. Experimental study of pain in human biliary tract induced by spasm of sphincter of Oddi. *Surg., Gynec. & Obst.*, 1940, *70*, 18–24.
31. LEVEN, N. L. Reflux into major pancreatic duct during cholangiography. *Proc. Soc. Exper. Biol. & Med.*, 1938, *38*, 808–809.
32. LONG, H. Observations of choledochoduodenal mechanism and their bearing on physiology and pathology of biliary tract. *Brit. J. Surg.*, 1942, *29*, 422–437.
33. LUETH, H. C. Studies of flow of bile into duodenum and existence of sphincter of Oddi. *Am. J. Physiol.*, 1931–1932, *99*, 237–252.
34. LÜTKENS, U. Aufbau und Funktion der extrahepatischen Gallenwege mit besonderer Bezugnahme auf die primären Gallenwegstauungen und die Gallensteinkrankheiten. F. C. W. Vogel, Leipzig, 1926.
35. McGOWAN, J. M., and BAKER, J. O. Relation of pregnancy to biliary disease and control of vomiting of pregnancy. *Canad. M. A. J.*, 1938, *39*, 133–137.
36. McGOWAN, J. M., BUTSCH, W. L., and WALTERS, W. Pressure in common bile duct of man; its relation to pain following cholecystectomy. *J.A.M.A.*, 1936, *106*, 2227–2230.
37. McGOWAN, J. M., KNEPPER, P. A., WALTERS, W., and SNELL, A. M. Relation of spasm of second portion of duodenum to biliary colic. *Surg., Gynec. & Obst.*, 1938, *66*, 979–987.
38. MANN, F. C., and GIORDANO, A. S. *Arch. Surg.*, 1923, *6*, 1.
39. MIRIZZI, P. L. Physiologic sphincter of the hepatic bile duct. *Arch. Surg.*, 1940, *41*, 1325–1333.
40. MIRIZZI, P. L. Functional disturbance of choledochus and hepatic bile ducts. *Surg., Gynec. & Obst.*, 1942, *74*, 306–318.

41. Moulonquet, P., and Malgras, P. L'exploration radiologique post operatoire de la voie biliaire principale. Masson & Cie, Paris, 1938, p. 41.
42. Negri, A. Exploración funcional de la porción terminal del conducto biliar. *Prensa méd. argent.*, 1945, *32*, 237–245. Consideraciones sobre el mecanismo del reflujo bilio pancreático. *Prensa méd. argent.*, 1945, *32*, 994–1002.
43. Newman, C. Physiology of gall-bladder and its functional abnormalities. *Lancet*, 1933, *1*, 785; 841; 896.
44. Nuboer, J. F. Studien über des extrahepatische Gallenwegssystem. *Frankfurt. Ztschr. f. Path.*, 1931, *41*, 454–511.
45. Opie, E. L. *Bull. Johns Hopkins Hosp.*, 1903, *14*, 229.
46. Pribram, B. O. New methods in gall-stone surgery. *Surg., Gynec. & Obst.*, 1935, *60*, 55–64.
47. Rienhoff, W. F., Jr., and Pickrell, K. L. Pancreatitis; anatomic study of the pancreatic extrahepatic biliary system. *Arch. Surg.*, 1945, *51*, 205–219.
48. Sandblom, P. *Svenska läk.-tidning*, 1932, *50*.
49. Sandblom, P., Voegtlin, W. L., and Ivy, A. C. Effect of cholecystokinin on choledochoduodenal mechanism (spincter of Oddi). *Am. J. Physiol.*, 1935, *113*, 175–180.
50. Schirmer, A. M. Beitrag zur Geschichte und Anatomie der Pankreas. *Inaug. Diss.*, Basel, 1893.
51. Schöndube, W. Ueber das Röntgenbild der kranken steinfreien Gallenblase und einige neue röntgenologische Beobachtungen über die Kinetik der extrahepatischen Gallenwege. *Verhandl. d. deutsch. Gesellsch. f. inn. Med.*, Kong. 44, 1932, pp. 381–389.
52. Schrager, V. L., and Ivy, A. C. Symptoms produced by distention of gallbladder and biliary ducts; clinical and experimental study. *Surg., Gynec. & Obst.*, 1928, *47*, 1–13.
53. Schwegler, R. A., Jr., and Boyden, E. A. Development of pars intestinalis of common bile duct in human fetus, with special reference to origin of ampulla of Vater and sphincter of Oddi; composition of musculus proprius. *Anat. Rec.*, 1937, *68*, 193–219.
54. Snell, A. M., McGowan, J. M., and Butsch, W. L. Colics following cholecystectomy; probable mechanism of their production. *Rhode Island Med. J.*, 1936, *19*, 113–119.
55. Sussman, M. L. Emptying of normal gallbladder. Am. J. Roentgenol. & Rad. Therapy, 1937, *38*, 867–871.
56. Walters, W., and Snell, A. M. Diseases of the Gallbladder and Bile Ducts. W. B. Saunders Co., Philadelphia, 1940, pp. 140, 376.
57. Weir, J. F., and Snell, A. M. Symptoms that persist after cholecystectomy; their nature and probable significance. *J.A.M.A.*, 1935, *105*, 1093–1098.
58. Westphal, K. *Ztschr. f. klin. Med.*, 1923, *96*, 22.
59. Westphal, K., Gleichmann, F., and Mann, W. Gallenwegsfunktion und Gallensteinleiden. J. Springer, Berlin, 1931.
60. Westphal, K. Die Bewegungs- und Resorptionsstörungen an den Gallenwegen und ihre Gefahren. *Verhandl. d. deutsch. Cesellsch. f. inn. Med.*, Kong. 44, 1932, pp. 354–363.

SIMPLE SPONTANEOUS PNEUMOTHORAX IN APPARENTLY HEALTHY INDIVIDUALS

A REPORT OF TWENTY-FOUR CASES

By R. F. NIEHAUS, M.D.
SAN DIEGO, CALIFORNIA

INTRODUCTION

IN THE past it has been customary for the medical profession to place the stigma of tuberculosis on cases of simple spontaneous pneumothorax.

During the past decade, simple spontaneous pneumothorax has come to be recognized as a fairly common clinical entity, especially in apparently healthy young men. Studies of large groups of young individuals in schools, colleges, in the armed forces and industry have supplied the medical literature with an ever increasing number of case reports.

Simple (benign, idiopathic or temporary) spontaneous pneumothorax may be defined as pneumothorax occurring suddenly in otherwise healthy individuals. The collapse is accompanied by little or no fever; pleural effusion is infrequent and when present very small in amount. Often the affected lung re-expands in two to six weeks. In the majority of patients, recovery is complete and without residual ill effects. The other lung as well as the re-expanded lung shows no evidence of active pulmonary tuberculosis. The sputum is negative for tubercle bacilli and the tuberculin test is frequently negative. Finally, during subsequent years, active pulmonary tuberculosis develops in these patients no more frequently than in any other similar group.

This paper presents 24 case histories on two separate groups of patients having this condition. The first group, totaling 14 patients, was studied at the University of Iowa Hospitals and represents the number of cases discovered during a fifteen year period (1927–1942). The second group, totaling 10 patients, was seen during a nine year period (1934–1943) at the Rees-Stealy Clinic in San Diego, California.

CASE REPORTS

CASE 1 (33740). R.D., male, aged twenty-six. In February, 1927, this patient experienced a sudden sharp pain in his left lower chest. The attack of pain occurred while he was lifting a large piece of lumber. The chest pain was intensified by taking a deep breath. He also complained of shortness of breath. The patient was seen by his local physician who made a diagnosis of pleurisy and immobilized his chest with adhesive tape. There was a low grade fever of 99.5° F. He remained at home and rested. Two months after the onset of his illness he was seen at the University of Iowa Hospitals. A chest roentgenogram taken on April 20, 1927, demonstrated a complete left-sided pneumothorax.

A follow-up letter in March, 1941, revealed that the patient had a recurrent attack of spontaneous pneumothorax involving the left side during the summer of 1940. This recurrent attack was thirteen years after his initial lesion. There was no evidence of pulmonary tuberculosis on the follow-up chest roentgenograms.

CASE 2. W.H., female, aged twenty-three. In May, 1942, this patient, a student nurse, awakened suddenly because of a "sticking pain" in the right chest. The patient discovered that the pain was eased by lying on her left side. Two hours later she again awakened and noted that the pain in the right chest was more severe and was associated with dyspnea. No cough was experienced. There had been no previous physical exertion. The patient was examined by a staff hospital physician who found clinical evidence of a right-sided pneumothorax. A chest roentgenogram taken on May 12, 1942, revealed a 100 per cent collapse of the right lung with displacement of the mediastinal structures to the left side. There was no free pleural fluid. The patient was hospitalized for fifty-eight days and was kept at bed rest for the major part of her stay in the hospital. Complete re-expansion of the right lung took place by July 14, about two months after the onset of the pneumothorax. Sputum examination was negative for

tubercle bacilli on two occasions in June, 1942. A Mantoux test was also negative. The patient returned to duty as a nurse in August, 1942, or in about three months after the onset of the pneumothorax. A roentgenogram of the lungs taken on June 8, 1944, about two years after the pneumothorax, showed no evidence of tuberculosis.

Case 3 (74196). W.J.W., male, aged thirty-one. While raising his arm to open a car door, this patient experienced a sudden, severe pain in his left chest. Shortness of breath accompanied this attack. A chest roentgenogram taken on December 12, 1932 (six days after the onset) demonstrated a complete collapse of the left lung with a small amount of pleural fluid at the left base. Re-expansion took place at the end of one month. Patient returned to the University of Iowa Hospitals for a physical checkup on March 31, 1934 (fourteen months after the onset of the left-sided spontaneous pneumothorax). He volunteered no complaints. However, a chest roentgenogram at this time demonstrated a partial collapse of the right lung. At the end of three months there was complete absorption of the right-sided pneumothorax. This patient could not be located by letter which was sent out on February 21, 1941.

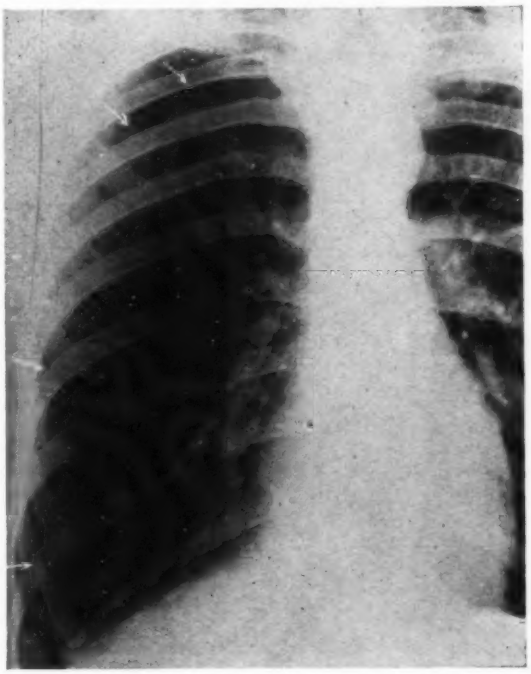


Fig. 1. Case 5. Partial spontaneous pneumothorax of the right lung. April 12, 1934.

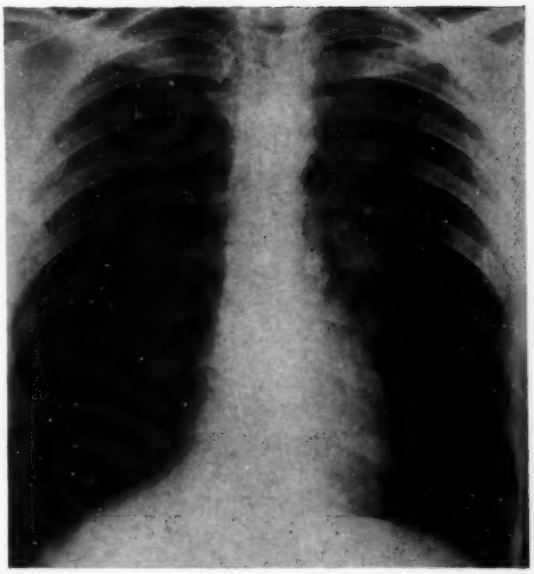


Fig. 2. Case 5. Re-examination one month later reveals complete expansion of the right lung. This patient has had a yearly roentgen examination of the lungs for the past seven years. There has been no recurrence of spontaneous pneumothorax and no evidence of active pulmonary tuberculosis.

Case 4 (80259). T.F., male, aged twenty-three. On September 19, 1933, this patient had a sudden onset of pain in his mid-chest just to the right of the sternum. The pain was aggravated by deep breathing. A chest roentgenogram demonstrated a partial collapse of the right lung. Re-expansion of the affected area was complete one month later.

Three months later this patient had a recurrence of his right-sided spontaneous pneumothorax. His history on the second admission to the University of Iowa Hospitals revealed right-sided pleural pain of ten days' duration. The patient (a physician) had also noted a pleural friction rub on the right side. A roentgenogram taken on December 18, 1933, again showed a partial collapse of the right lung. Complete absorption of this pneumothorax occurred at the end of five weeks. A follow-up letter sent out on February 21, 1941 (more than seven years after the onset of the initial attack) revealed no further recurrence of a spontaneous pneumothorax and there had been no evidence of pulmonary tuberculosis.

Case 5 (84602). E.L., male, aged twenty-three. The only available history of this patient is that of right-sided pleural pain of two days'

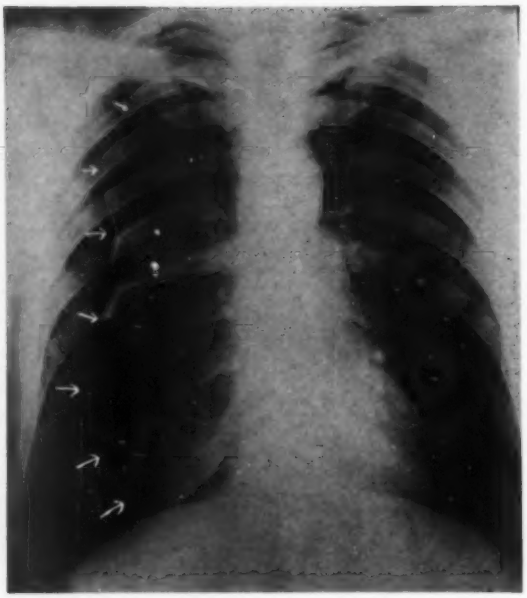


FIG. 3. Case 6. Partial spontaneous pneumothorax of the right lung. June 23, 1936. This patient has had adequate follow-up examinations during the past five years. There has been no recurrence of spontaneous pneumothorax and no evidence of active pulmonary tuberculosis.

duration. On April 12, 1934, chest roentgenograms revealed a partial collapse of the right lung. Progress roentgenograms showed complete re-expansion of the right lung one month later. A follow-up letter sent to this patient on February 21, 1941, revealed that he had had yearly physical examinations and chest roentgenograms since the onset of his spontaneous pneumothorax. During this seven year follow-up period there had been no recurrence of spontaneous pneumothorax and no evidence of pulmonary tuberculosis.

CASE 6 (103355). W.A.B., male, aged thirty-three. On June 21, 1935, this patient noted a sudden, sharp pain in the right side of his chest. He immediately went home to bed but the chest pain persisted throughout the night. It was aggravated by moving about and by coughing. There was no elevation of temperature. The following day a roentgenogram of his chest demonstrated partial collapse of the right lung. A follow-up letter written February 20, 1941 (almost five years later) revealed no recurrence of the spontaneous pneumothorax and there had been no evidence of pulmonary tuberculosis.

CASE 7. M.O., female, aged thirty-two. Patient was awakened from her sleep on July 30, 1936, by an acute knife-like pain in the left chest. The pain was made worse by body movement and upon taking a deep breath. There was no cough or elevation of temperature. The physical findings were those of a pneumothorax. A chest roentgenogram taken on the following day demonstrated a partial pneumothorax on the left side. Re-expansion of the partially collapsed lung took place one week later. This patient is employed as a nursing supervisor at the University of Iowa Hospitals. Personal communication with this patient revealed that there had been no recurrence of spontaneous pneumothorax during the past five years. Yearly chest roentgenograms have shown no evidence of pulmonary tuberculosis.

CASE 8 (105522). D.V., male, aged twenty-eight. This patient experienced a sudden onset of right-sided pleural pain and shortness of breath on September 10, 1936. There had been no previous physical exertion. Physical examination revealed typical findings of pneumothorax. A chest roentgenogram taken on September

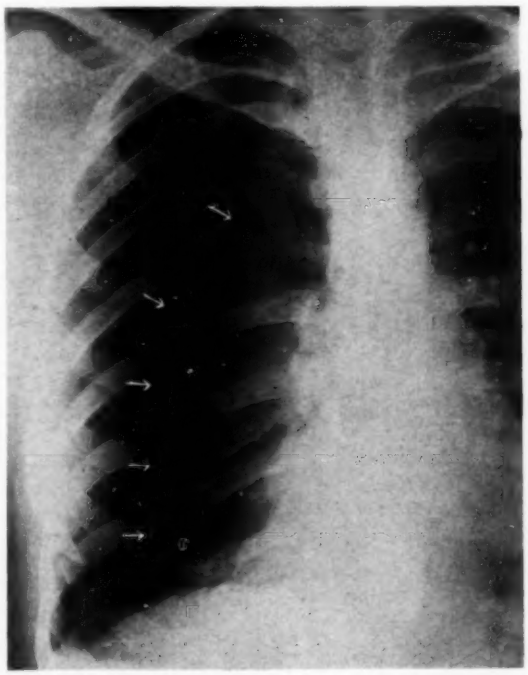


FIG. 4. Case 10. Almost complete spontaneous pneumothorax of the right lung. There is a small amount of fluid in the right costophrenic sinus. June 20, 1938.

15, 1936, revealed an almost complete collapse of the right lung. There was complete re-expansion of the affected lung at the end of nine weeks. A follow-up letter four and one-half years after the onset of his attack revealed no recurrence of spontaneous pneumothorax and no evidence of pulmonary tuberculosis.

Case 9 (118773). W.J., male, aged nineteen. On March 3, 1938, this patient "blocked" a fellow player during football practice. His breath was "knocked out" of him momentarily, but shortly thereafter he continued playing. He felt perfectly well for the next three days. But on March 7, 1938, he complained of shortness of breath, a disturbing unproductive cough and pain in his right chest. A chest roentgenogram taken at this time demonstrated a complete collapse of the right lung. There were no fractured ribs. Progress chest roentgenograms revealed complete re-expansion of the right lung at the end of four weeks. On January 24, 1941 (approximately two years and ten months later) the patient had a recurrence of his right-sided spontaneous pneumothorax. His symptoms at this time were those of a mild upper respiratory infection accompanied by severe pleural pain on the right side. Chest roentgenograms have shown no evidence of pulmonary tuberculosis in almost three years of observation.

Case 10 (121711). L.V.R., female, aged twenty-three. This patient gave a history of

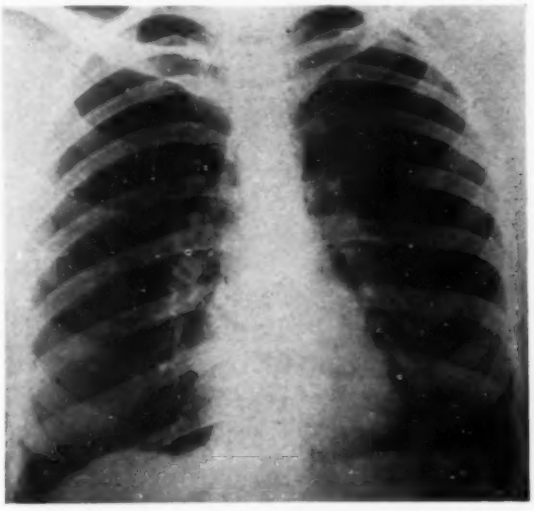


Fig. 5. Case 10. Complete re-expansion of the right lung one month later, July 20, 1938.

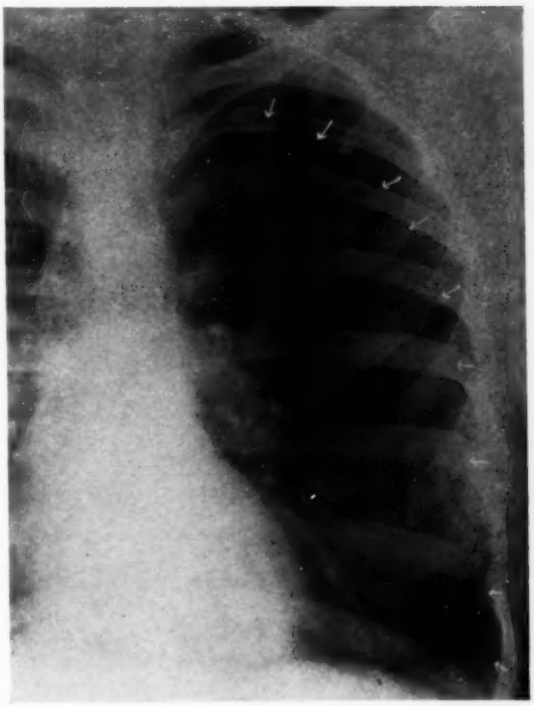


Fig. 6. Case 10. Partial spontaneous pneumothorax of the left lung. April 3, 1939. (Approximately ten months after the right-sided pneumothorax.)

mild, recurrent attacks of pleurisy over a ten year period. Then on the night of June 19, 1938, while talking to friends she suddenly experienced a sharp pain high in the right chest. The pain radiated down the right arm. Coughing, shortness of breath and mild shock accompanied the chest pain. Physical examination revealed the typical findings of a pneumothorax. A chest roentgenogram on June 20, 1938, showed a partial collapse of the right lung with a small amount of fluid in the right costophrenic sinus. Re-expansion of the affected lung occurred one month later.

On April 3, 1939 (almost ten months later) this patient developed sudden, sharp pain in the left axilla which radiated to the left shoulder and neck. This occurred while stooping over to pick up an object from the floor. There was some associated shortness of breath. A roentgenogram on the same day showed a partial pneumothorax of the left lung. Complete absorption of this left-sided pneumothorax took place at the end of three weeks. A follow-up letter revealed there had been no recurrence of spontaneous pneumothorax or evidence of pul-

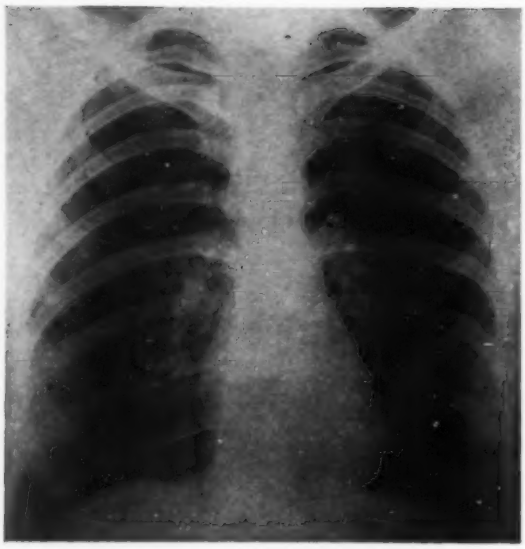


FIG. 7. Case 10. Complete re-expansion of the left lung took place three weeks later—April 24, 1939. There has been no evidence of active pulmonary tuberculosis during a twenty-two month follow-up period.

monary tuberculosis during a twenty-two month period.

CASE 11 (125147). D.S., male, aged twenty-six. On September 30, 1938 (two weeks prior to admission to the University of Iowa Hospitals) this patient suddenly developed a sharp, almost continuous pain in the left chest at the level of the fifth interspace. The pain was intensified by coughing and it radiated posteriorly to the spine and anteriorly to the sternum. Six days prior to his hospitalization he experienced shortness of breath which prevented his lying flat in bed. There was no chill, fever, or cyanosis. He had always enjoyed excellent health prior to this illness. Physical examination on October 14, 1938, revealed the typical findings of pneumothorax. A chest roentgenogram taken at this time demonstrated partial collapse of the left lung. In addition, a large bullous bleb measuring 3 by 5 cm. was noted in the apex of the right lung. The left-sided pneumothorax was completely absorbed six weeks. later. A roentgen examination of the lungs in October, 1939, about thirteen months after the pneumothorax showed no evidence of pulmonary tuberculosis.

CASE 12 (133089). J.G., male, aged forty-six. While hauling coal, this patient had a sudden onset of severe, choking pain in the right lower chest which radiated to the right shoulder. Shortness of breath accompanied this attack. He was admitted to a hospital at Council Bluffs, Iowa, where a roentgenographic examination demonstrated a complete collapse of the right lung. The patient remained in bed for five weeks and was then sent to the University of Iowa Hospitals on August 11, 1939. A roentgenogram taken on the day of hospital admission revealed a partial pneumothorax of the right lung. At the end of six weeks there had been complete re-expansion of the affected lung. There has been no recurrence of spontaneous pneumothorax or active pulmonary tuberculosis during a twenty-one month follow-up period.

CASE 13 (138568). J.J.C., male, aged twenty-two. On February 27, 1940, this patient developed a sudden knife-like pain in the left chest immediately following the throwing of a snowball. The pain extended from the left scapula to the anterior chest wall. This was accompanied by shortness of breath. A roentgenogram of the chest taken on the same day demonstrated a partial collapse of the left lung.

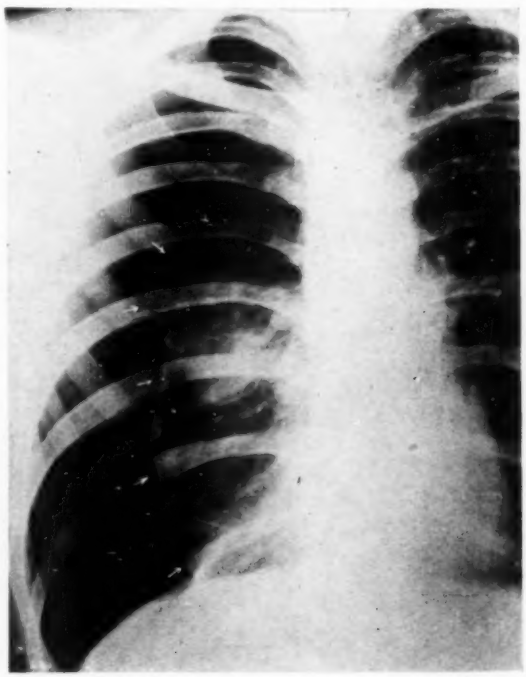


FIG. 8. Case 14. Almost complete spontaneous pneumothorax of the right lung. January 27, 1941.

Progress roentgenograms revealed complete absorption of the partial pneumothorax seven days after the onset. A follow-up chest roentgenogram taken one year later showed no evidence of tuberculosis.

Case 14 (140531). J.P., male, aged twenty-two. This patient gave a history of awakening on the morning of January 26, 1941, with a "pleurisy" in the right chest. This was accompanied by shortness of breath. In addition, it was noted that the right hemithorax was enlarged. There were physical signs of a pneumothorax. Chest roentgenograms taken on February 27, 1941, showed an almost complete collapse of the right lung. There was complete re-expansion of the affected lung at the end of four weeks. The patient, a medical student, returned to class work about two months after the onset of the pneumothorax. The last follow-up roentgen examination of the lungs taken in 1942, about one year after the pneumothorax, showed no evidence of pulmonary tuberculosis.

Case 15 (47900). R.L.B., male, aged twenty-four. On December 7, 1940, this patient complained of a sudden, sharp pain in the right lower chest. There was associated mild shortness of breath and an unproductive cough. No preceding exertion or trauma had taken place.

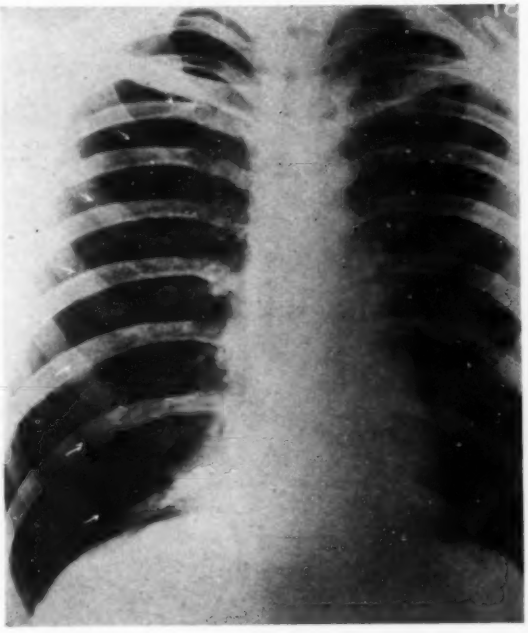


Fig. 9. Case 14. Partial re-expansion of the right lung about one month later, February 21, 1941.

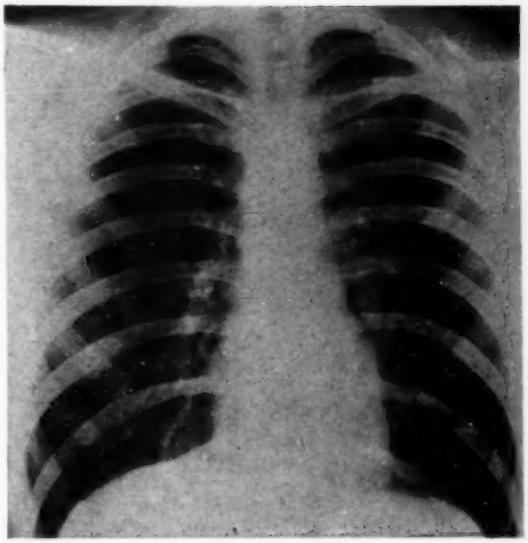


Fig. 10. Case 14. Complete re-expansion of the right lung took place at the end of five weeks. This patient, a medical student, has shown no evidence of active pulmonary tuberculosis during a twelve month follow-up period.

The family physician strapped his chest and gave him a sedative. The following day roentgenoscopy revealed approximately 70 per cent collapse of the right lung. A small amount of fluid was noted in the right costophrenic angle. The patient was placed at bed rest for sixteen days and allowed to return to work as a clerk on December 30. On January 13, 1941, thirty-seven days after the onset of the spontaneous pneumothorax, a roentgen examination revealed complete re-expansion of the collapsed lung. There was no evidence of active parenchymal tuberculosis. In January, 1945, about four years and one month after the onset of his spontaneous pneumothorax, the patient is reported to be in good health.

Case 16 (59548). W.P.J., male, aged forty-two. On June 10, 1942, this patient, in running to answer the telephone, slipped on an oil-covered floor. He landed in an awkward sitting position with both arms fully extended overhead. He immediately experienced sharp pain at the top of his right chest in the region of the clavicle. He did not consult a physician on the day of the accident. A dull chest ache persisted throughout the night and interfered with sleep. The following day he drove his car 150 miles and reported before an examining board for a physical examination for the armed forces. On

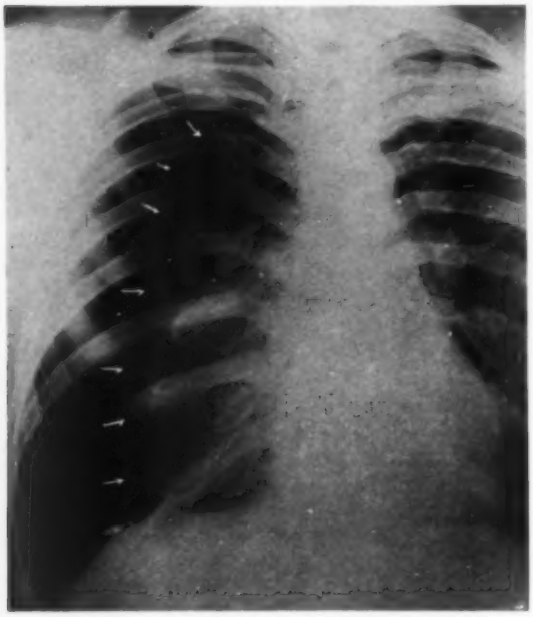


FIG. 11. Case 17. Partial spontaneous pneumothorax of the right lung. October 23, 1942.

returning home he experienced marked "'pressure pain" in the right chest. There was associated shortness of breath. He found it necessary to drive most of the way with his left hand and used the right hand to splint his chest. The patient consulted his family physician three days after the accident. His physician found a 70–80 per cent collapse of the right lung. A trace of fluid was noted in the right costophrenic angle; 850 cc. of air was aspirated from the right chest followed by relief of the "pressure pain." Patient was hospitalized for about seven days. On July 16, 1942, about thirty-six days after the the onset of the spontaneous pneumothorax, a progress roentgenogram revealed complete re-expansion of the right lung. No evidence of active parenchymal tuberculosis was demonstrated. The patient returned to work on August 1, 1942, about six weeks after the onset of the pneumothorax. In January, 1945, about two years and eight months after the onset of the pneumothorax, the patient is reported to be working and in good health.

CASE 17 (61388). L.L.O., male, aged twenty-seven. On October 15, 1942, this patient, a city fireman, was lifting a suction hose and connecting it to a fire apparatus. The patient was in an awkward, stooping position and experienced a sudden sharp pain in his right chest. There was associated shortness of breath. A physician was immediately consulted and roentgenoscopic examination revealed an almost 100 per cent collapse of the right lung. On November 30, 1942, forty-one days later, follow-up roentgen examination revealed the right lung to have fully re-expanded. There was no evidence of active parenchymal tuberculosis. The patient returned to duty on December 7, 1942, approximately seven weeks after the onset of the pneumothorax. In January, 1945, about two years and three months after the spontaneous pneumothorax, the patient is reported to be working and in good health.

CASE 18 (72545). W.M., male, aged twenty. On December 1, 1943, this patient, while walking, experienced a dull pain in his left chest which increased in severity over a period of several hours. He consulted a family physician who prescribed a sedative. The left chest pain persisted and several days later his family physician ordered a roentgenogram of the chest. A 25 per cent collapse of the left lung was demonstrated. A roentgenogram taken on December 15, 1943, fourteen days after the onset of the attack, revealed complete re-expansion of the collapsed lung. There was no

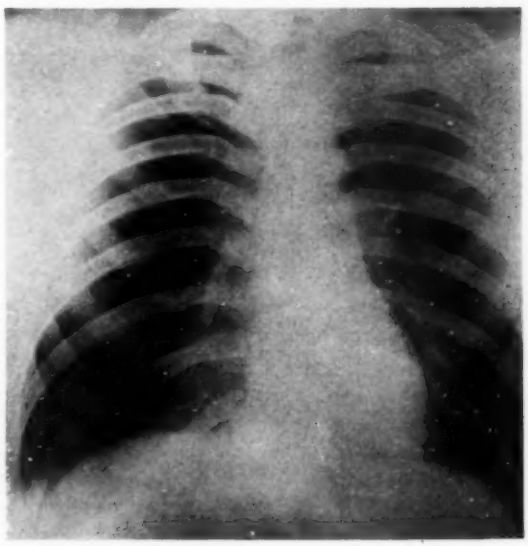


FIG. 12. Case 17. Complete re-expansion of the right lung about four weeks later, November 20, 1942. This patient, a city fireman, returned to duty seven weeks after the onset of the pneumothorax. There has been no evidence of active pulmonary tuberculosis during twenty-seven month follow-up period.

evidence of active parenchymal tuberculosis. An intradermal tuberculin test was reported to be 1 plus. In January, 1945, about two years and one month after the spontaneous pneumothorax, the patient is reported to be working and in good health.

Case 19 (46758). J.D., male, aged twenty-one. On the evening of March 31, 1943, this patient suddenly developed sharp precordial pain followed by a severe coughing attack and associated shortness of breath. There had been no preceding physical exertion. Roentgenoscopic examination the next day revealed a 60 per cent collapse of the left lung. A roentgen examination on May 27, 1943, approximately two months after the spontaneous pneumothorax, demonstrated a re-expansion of the collapsed left lung. There was no evidence of active parenchymal tuberculosis. In January, 1945, about one year and ten months after the spontaneous pneumothorax, the patient is reported to be working and in good health.

Fig. 13. Case 20. This patient had recurrent left-sided attacks of spontaneous pneumothorax over a period of two years. This roentgenogram was taken on April 28, 1943, about three months after the induction of a left-sided chemical pleuritis. The left lung has fully re-expanded and has remained so over a two year follow-up period. The obliterated left costophrenic angle is the end-result of the chemical pleuritis. There has been no evidence of pulmonary tuberculosis.

Case 20 (58988). J.F., male, aged thirty-nine. On December 22, 1942, this patient experienced severe, sharp pain in the left chest with associated "pressure sensation," dyspnea and cough. Physical examination at this time revealed diminished breath sounds over the entire chest with tympany on percussion. A roentgen examination revealed a 100 per cent collapse of the left lung. Air was aspirated from the left chest on three occasions: December 28, 1942, January 11 and 30, 1943, and relief of the pressure symptoms was obtained.

The patient had had a spontaneous collapse of the left lung on December 8, 1941, almost one year prior to the present attack. Following this initial collapse, the left lung was reported to have fully re-expanded after bed rest period of seven weeks.

Because of the history of recurrent spontaneous pneumothorax, a 5 cc. mixture of mineral oil and 1 per cent gomenol was introduced into the left pleural space on January 23, 1943 (about one month after the present spontaneous collapse). Three months after the induction of a chemical pleuritis, the left lung had fully re-expanded. In January, 1945, about two years after the induction of a chemical pleuritis, the patient is reported to be working and in good health.

Case 21 (38314). G.L.W., male, aged thirty-eight. On October 26, 1938, while sitting at work, this patient felt a sharp pain in the left chest, associated with shortness of breath. He had just completed some emergency repair work on an airplane. A roentgen examination taken about one week after this attack revealed an almost complete collapse of the left lung.

The patient's past medical history revealed that he had experienced spontaneous collapse of the left lung in October, 1934, four years prior to the present attack. The left lung was reported to have re-expanded within two months under conservative medical management.

Because of the history of recurrent attacks of spontaneous pneumothorax, 5 cc. of lipiodol was introduced into the left pleural cavity on November 16, 1938, (twenty-one days after the present attack). On March 16, 1939, approximately four months after the induction of a chemical pleuritis, the left lung was reported to be completely re-expanded. A roentgenogram of the lungs taken on July 27, 1940, nine months after the second attack, revealed no

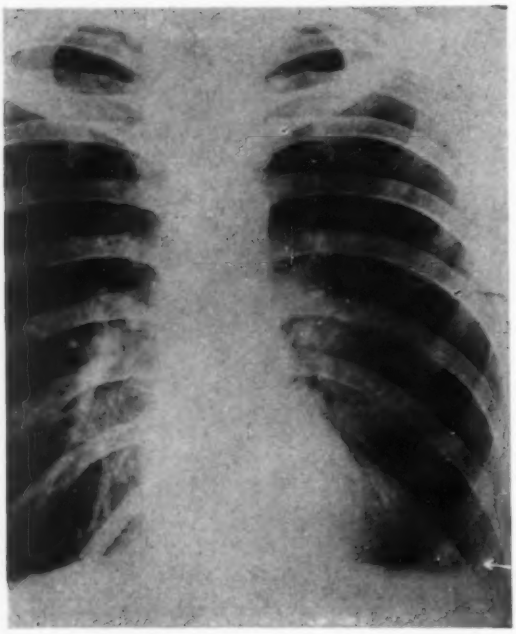


FIG. 14. Case 21. This patient had recurrent attacks of left-sided spontaneous pneumothorax over a five year period. This roentgenogram was taken on March 16, 1939, about four months after the induction of a chemical pleuritis. The left costophrenic angle is obliterated. The patient is reported to be working and in good health six years and two months after the induction of the chemical pleuritis.

active parenchymal tuberculosis. A minimal thickening at the left base was demonstrated and it was thought to have been the result of the chemical pleuritis. In January, 1945, about six years and two months after the production of an aseptic pleuritis, the patient is reported to be working and in good health.

CASE 22 (29612). W.A.T., male, aged twenty-five. This patient, a Lieutenant in the U. S. Navy Air Force, consulted a physician on November 30, 1936, because he thought he had a collapsed lung. About three months previously while pulling out of a power dive, he experienced a sudden sharp pain in the right lower chest. There was no accompanying shortness of breath. The sharp pain lasted only a few days but the patient had been aware of a "clicking sound" in the chest for the past few weeks.

On physical examination on November 30, 1936, there was suggestion of increased resonance and slightly distant breath sounds over the right lower lobe posteriorly. A roentgenogram of the lungs revealed a partial pneumothorax of the right lower lobe. Two weeks later the patient informed his physician that he thought his lung had undergone further collapse. The patient admitted that he had not taken bed rest as instructed. Instead, he had been inflating toy balloons in an attempt to bring about re-expansion of the collapsed lung. Roentgenoscopic examination demonstrated about 35 per cent collapse of the lower two-thirds of the right lower lung field. Three weeks later a roentgenogram of the lungs demonstrated about a 50 per cent collapse of the right lower lung field. The patient was placed in a naval hospital and under conservative management the collapsed lung failed to re-expand over a ten month period. In October, 1937, a thoracotomy was performed and the visceral pleura was sutured to the chest wall at the site of the pleuropulmonary communication. The patient

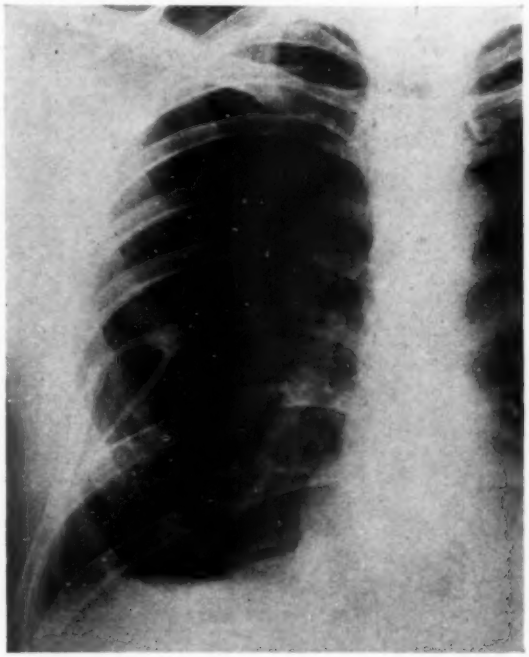


FIG. 15. Case 22. This patient, a Naval flier, had recurrent attacks of right-sided spontaneous pneumothorax over an eighteen month period. In October, 1937, a rib resection was done and the visceral pleura was sutured to the chest wall at the site of the pleuropulmonary communication. This roentgenogram was taken one year after the rib resection (October, 1938). The patient returned to active duty early in 1938 and is now reported to be overseas.

returned to active duty early in 1938 and is now reported to be overseas.

Case 23 (54561). P.M., male, aged twenty-five. This patient noted sudden sharp pain in the right chest while playing baseball on March 15, 1932. He also experienced some shortness of breath. Physical examination revealed hypersonance with amphoric breath sounds over entire right chest. A roentgenogram taken on March 17, 1932, two days after his chest pain, revealed a complete collapse of the right lung. Roentgenoscopic examination on April 8, 1932, twenty-one days after the spontaneous pneumothorax, revealed complete expansion of the right lung. There was no evidence of active parenchymal tuberculosis. In February, 1945, about twelve years after the spontaneous pneumothorax, the patient is reported to be working and in good health.

Case 24 (5400). A.E.S., female, aged thirty-six. This patient was first seen on August 10, 1934, for a physical checkup. She complained of some shortness of breath but no chest pain. A roentgen examination demonstrated almost complete collapse of the right upper lobe. On September 6, 1934, about twenty-seven days later, the collapsed lobe of the lung had completely re-expanded. A progress roentgen examination of the lungs showed no evidence of active parenchymal tuberculosis. On May 14, 1936, about twenty months after the first attack, the patient complained of substernal chest pain and a non-productive, spasmodic cough. Roentgenoscopic examination revealed a 100 per cent collapse of the right lung field. Periodic examination at monthly intervals revealed a very slow expansion of the affected lung. Complete re-expansion of the right lung did not take place until April 30, 1937, almost one year later. This patient had a coronary occlusion in 1944, about seven years after the recurrent spontaneous pneumothorax. In February, 1945, about ten years and three months after the initial spontaneous pneumothorax, she is reported to be in fairly good health.

INCIDENCE

Kjaergaard[8] found 51 case records of simple spontaneous pneumothorax in the hospitals of Denmark covering a twenty year period. Perry[14] has reported 85 cases at the London Hospital over a fourteen year period. Blackford[1] cited 15 cases found in the student body at the University of Virginia. Eleven of the 15 cases occurred in a five year period. Wilson[21] reported on 11 cases during a five year period among male students at Yale University. Ornstein and Lercher[12] reported on their experiences with 58 cases of this condition seen in private practice during a twenty year period. Eight hundred and seventy-three hospital admissions with a primary diagnosis of simple spontaneous pneumothorax were recorded in the Army in the continental United States in 1943. In about 15 per cent of the soldiers concerned, the disability was considered of sufficient gravity to warrant separation from the service. The remainder, all of whom were returned to general or restricted duty, averaged about forty days in the hospital.

Examination of the 24 patients reported in this paper reveals a striking constancy in the age and sex incidence of this condition. Twenty of the 24 cases were males and 21 of the 24 cases occurred in persons between the ages of twenty and forty years. One of the 3 remaining cases was nineteen years of age and 2 others were between the ages of forty and forty-five. The average age for this group of 24 patients was twenty-eight years and two months.

This condition involved the right side of the chest slightly more than the left; 15 of the 24 cases involved the right side, and 9 cases involved the left side. This difference is probably not significant.

PATHOGENESIS

From all the present findings, both clinical and anatomical, Kjaergaard[8] maintains that simple spontaneous pneumothorax is due to rupture of a valve vesicle anywhere on the surface of the lung. The valve vesicles may be either a scar tissue vesicle, an emphysematous valve vesicle or a congenital valve vesicle. Up to 1933 there were 8 autopsy records in which this condition had been fully established. Three of them were cases with scar tissue vesicles (Hayaski[5] 2 cases, Fisher[3]), three others with emphysematous valve vesicles (Ranking,[16]

Pitt,[15] Brunner[2]), and Kjaergaard[9] reported 2 cases having congenital valve vesicles.

It is believed that all three types of subpleural blebs or vesicles have a valve-like structure at their pleural attachment. Moreover, Kjaergaard[8] thinks that this valve-like flap would allow air to pass from a terminal bronchiole into the vesicle, but prevent it from passing out again. Since a valve-like mechanism is thought to be basically similar in all three types of subpleural vesicles, only the scar tissue vesicle will be described in detail.

FIG. 16. Emphysematous valve vesicle. The outline of the vesicle is indicated by the dotted line. (After Kjaergaard.[8])

The scar tissue vesicle is usually located at the apex of the lung. As to the origin of the scar tissue vesicle, neither Hayaski[5] nor Fisher[3] is able to offer any definite conclusion. Two of their 3 autopsied cases showed no signs of tuberculosis either grossly or microscopically. Nevertheless, these authors consider a previous, limited, healed tuberculous lesion to be the most probable cause. The physical basis for the formation of the scar tissue bleb or vesicle is thought to be due to scarring close to the visceral pleura with resultant localized emphysema and distortion of the terminal bronchioles. Microscopic examination of these vesicles by Fisher[3] and Hayaski[5] showed a valve-like structure at their bases. These valves simulated the flap valve in an ordinary pump and consisted of tongue-like deformed atrophic lung tissue. The valve allowed air from a terminal bronchiole to pass freely into the vesicle, but prevented it from passing out again. Consequently, the vesicle gradually increased in size and a positive pressure was built up by respiratory movement. As a result, the pleural wall of the vesicle became progressively thinner and atrophic and finally rupture took place when the pressure was suddenly increased (as in coughing or sneezing) or during ordinary, quiet respiration.

Both Hayaski[5] and Kjaergaard[8] have searched for and found unruptured valve vesicles in a series of necropsies on patients dying of other causes. In these cases the gross and microscopic findings were identical to those with spontaneous collapse of the lung.

Kjaergaard[8] thinks it impossible for a normal pleura to rupture through coughing or by muscular exertion.

Experiments with inflation of the lungs at the autopsy table revealed that a rupture of the pleura could not be produced until the pressure was above 200 mm. Hg. It is believed that such a high intrapulmonary pressure can never occur in life.

Kjaergaard[8] also points out that patients with bullous emphysema of the lungs seldom have spontaneous pneumothorax. He has observed that these blebs or vesicles do not have a valve-like structure at their bases; but communicate freely with the underlying lung tissue. As a result, there is no tendency to build up a positive pressure within the bleb or vesicle, and hence conditions favorable for rupture do not exist.

In view of the foregoing, the valve is important both in relation to the formation of the pleural bleb and the mechanism of its rupture. Wilson[21] believes "the presence of valve vesicles explains the marked

tendency of spontaneous pneumothorax to recur. Also, since such vesicles are apt to be found in the apices of both lungs, if present in one, it explains the occurrence of bilateral simultaneous, or alternating pneumothoraces."

CLINICAL PICTURE

Symptomatology and Physical Signs. The onset of symptoms is usually abrupt. The usual initial complaint is pain, or, as many patients described it, "a sudden stitch in the chest." Shortness of breath and rapid pulse often accompany the chest pain. The symptoms vary ordinarily in proportion to the degree of collapse and to the displacement of the trachea and mediastinal structures. The pain is felt on the side of the pneumothorax and may be located anywhere from the shoulder to the upper abdomen. The temperature is rarely elevated. These symptoms usually subside in a few days. However, slight shortness of breath and tachycardia may remain if the affected lung is slow in re-expanding.

The physical signs are suppression of fremitus, breath and voice sounds. Inspection may reveal visible enlargement of the affected hemithorax with bulging of the costal interspaces. There may also be a slight lag in expansion on the affected side. Hyperresonance will be present where collapse is almost or entirely complete. Displacement of the heart toward the opposite side is found when total pneumothorax is reached.

Prognosis. In the vast majority of patients, the affected lung re-expands promptly in from two to six weeks. Recovery is complete and without residual ill effects. However, it is estimated that in about 10 to 20 per cent of cases, collapse of the lung does recur one or more times after short or very long intervals of time. Recurrent pneumothorax usually affects the same lung but occasionally the opposite lung is involved.

Nine, or about 27 per cent, of the 24 patients presented in this study suffered a recurrent pneumothorax. In 7 instances the collapse recurred on the same side. The remaining 2 patients experienced alternating spontaneous pneumothorax.

On rare occasions, simple spontaneous pneumothorax may become a persistent or chronic pneumothorax. This condition obtains when collapse of the lung persists for many months without the lung showing any or but slight evidence of re-expansion. None of the 24 cases presented in this paper could be classified as a persistent or chronic pneumothorax.

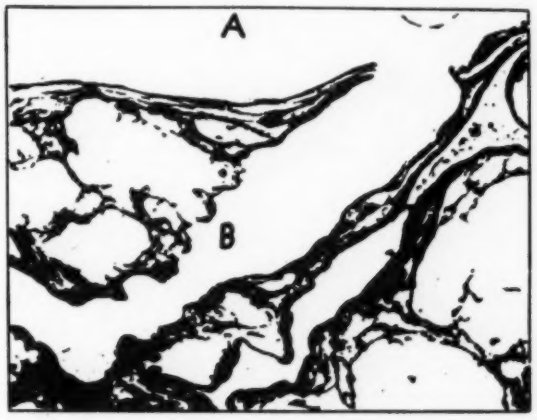


FIG. 17. Magnification of emphysematous valve vesicle shown in Figure 16. (*A*) lumen of vesicle; (*B*) respiratory bronchiole. The emphysematous lung tissue above the bronchiole forms a tongue-shaped flap which is thought to be the basis of the vesicle valve mechanism. (After Kjaergaard.[8])

It is only in rare instances that active pulmonary tuberculosis develops subsequent to attacks of spontaneous pneumothorax in healthy persons. In a survey of 49 subjects under observation for almost two years after their initial attack of pneumothorax, Kjaergaard[8] found but 1 case of active pulmonary tuberculosis and this patient had been recently exposed to an open case of pulmonary tuberculosis. Blackford[1] found that 14 out of 15 of his reported cases were known to be in good health after observation periods of eight months to fifteen years.

Twenty-one of the 24 patients in this study have remained in good health and have been followed adequately for periods

of one to twelve years. Two of the remaining 3 patients could not be located by mail. The third and last patient experienced a coronary occlusion approximately ten years after the pneumothorax and is still living.

Simple, uncomplicated spontaneous pneumothorax may recur once or several times, but the patient survives. However, there are certain rarer types of spontaneous pneumothorax in the healthy which should be mentioned because they involve danger to life. These are: (1) *Tension pneumothorax*. Tension pneumothorax is that form of spontaneous pneumothorax in which there is development of a high intrapleural pressure. It is reported in about 10 per cent of cases. This type is characterized by extreme dyspnea and cyanosis with marked mediastinal displacement toward the opposite side and the diaphragm on the affected side is pushed downward. The patient is in great respiratory and circulatory distress. The movement of the affected side is greatly restricted and the intercostal spaces on the affected side bulge. On physical examination the percussion note is hyperresonant, the breath sounds absent or greatly diminished and the liver dullness or precordial dullness disappears. Constitutional symptoms such as fever and tachycardia are often present. Death may come suddenly. The treatment of tension pneumothorax is one of repeated aspiration of sufficient quantities of air to relieve pressure on the mediastinal structure. In more severe cases, where a high intrapleural pressure is maintained, despite repeated aspiration of air, it may become necessary to place a needle between the ribs and permit the patient to "blow out" the air. (2) *Hemopneumothorax*. The clinical picture of spontaneous hemopneumothorax is that of an acute illness and the most striking and constant early symptom is pain. The initial pain is usually sudden in onset, sharp, stabbing and continuous in character and localized to the side of the chest which is involved. Hartzell[4] states that the pain not uncommonly radiates to the shoulder of the involved side, and, significantly, frequently radiates to the abdomen. Dyspnea usually accompanies the chest pain. Nausea, vomiting and even diarrhea are not uncommon. Shock, anemia, signs of collapse and internal hemorrhage follow in the more severe cases. The physical findings are those of pneumothorax with effusion.

Hartzell[4] reviewed 40 cases of spontaneous hemopneumothorax and added 3 of his own. Fourteen of these 43 patients died, 5 within twenty-four hours of hospital admission and 4 more within a week. Forty-two of the 43 patients were males. In all of Hartzell's cases, tuberculosis was adequately excluded as a cause of spontaneous collapse and associated bleeding. (3) *Bilateral simultaneous spontaneous pneumothorax*. Spontaneous pneumothorax of one lung with sudden change of intrathoracic pressure may lead to a collapse of the other lung. Death may take place before medical assistance can be secured. Continuous suction of air from both sides of the chest in the hope of partially re-expanding one or both lungs is indicated, coupled with the administration of oxygen. Perry[14] reviewed 20 cases of this condition and reports a 50 per cent mortality.

TREATMENT

In the treatment of simple spontaneous pneumothorax, the patient should be hospitalized promptly. If fever is present and dyspnea marked, absolute bed rest is imperative. The uncomplicated case requires but one to three weeks of bed rest since the collapsed lung often re-expands within a few weeks and occasionally in seven to ten days. Later, a period of light physical activity should be followed for at least one month. Straining at stool, lifting heavy objects, awkward positions, severe coughing attacks, should be avoided. Waring[20] also believes that ascent above 4,000 feet in an airplane should be forbidden. Re-expansion of the lungs should be checked by roentgenoscopic examination at appropriate intervals. The possibility of pulmonary tuberculosis must be ruled out. The sputum should be examined for

tubercle bacilli. A tuberculin skin test should be done. In addition, progress chest roentgenograms should be studied carefully for minimal tuberculous lesions both in the re-expanded lung and in the opposite lung.

Waring[20] suggests that occasionally small amounts of air (200–500 cc.) may be aspirated from the pneumothorax cavity at short intervals and re-expansion of the affected lung thereby hastened. However, this procedure must be undertaken cautiously for fear of enlarging the opening between the lung and pleura or of establishing a persistent pleuropulmonary communication or chronic pneumothorax.

The treatment of recurrent simple spontaneous pneumothorax is a separate problem. In this condition, the repeated attacks of a collapsed lung is both disabling and a financial burden to the patient. A simple procedure whereby recurrent pneumothorax may be avoided is therefore of importance. The induction of an aseptic or chemical pleuritis has been successfully used in a number of instances. Hennell and Steinberg[6] reviewed the literature and reported on 5 additional cases which were treated by chemical pleuritis. Some of the chemical irritants instilled into the pleural space for this purpose include hypertonic glucose solutions (30–67.5 per cent), hypertonic saline, 0.5–1.0 per cent silver nitrate, lipiodol, gomenol in olive oil and mineral oil. Particulate matter such as plain or iodized talc, powdered or in suspension, has also been used.

Two of the 24 patients presented in this study were treated by induction of a chemical pleuritis. Gomenol in mineral oil was used in 1 instance and lipiodol in the other case. Recurrent pneumothorax has not occurred in these 2 patients. Treatment was induced two years ago in the first instance and six years ago in the second case.

Waring[20] states that all chemical instillations are usually painful, and frequently accompanied by more or less severe though transient constitutional symptoms and pleural effusion. Moreover, Waring states that their ultimate effects are unpredictable.

There are also single case reports in which surgical intervention has been successfully used to prevent recurrent collapse of a lung. In cases in which roentgenograms or thoracoscopy demonstrates a single subpleural bleb or vesicle or pleural adhesion, surgical intervention (thoracotomy) to excise the bleb or sever the adhesion is recommended by some writers for the prevention of recurrent spontaneous pneumothorax. Tyson and Crandall[18] report a case of recurrent spontaneous pneumothorax in which a single bleb (5 cm. in diameter) was excised by thoracotomy. This patient has now been working three years and seven months with no symptoms or ill effects.

One of the patients in this present case study was also successfully treated by surgical intervention. This latter patient, a Lieutenant in the United States Naval Air Force experienced attacks of a spontaneous pneumothorax during a period of two years. In 1937, Eloesser performed a thoracotomy and successfully located and closed the pleuropulmonary communication and sutured the visceral pleura to the chest wall. The patient has been returned to active duty and is now reported to be overseas. It is presumed that this patient has remained in good health for a period of more than six years.

In the rare instances when simple spontaneous pneumothorax becomes a persistent or chronic pneumothorax, one is again faced with an individual problem. This condition obtains when there is a persistent pleuropulmonary communication which fails to heal under conservative medical management. Waring[20] recommends surgical intervention as the treatment of choice while Hennell and Steinberg[6] favor the induction of a chemical pleuritis.

SUMMARY

1. Simple spontaneous pneumothorax occurring in two groups of case studies

totaling twenty-four patients has been presented and the literature reviewed briefly. The condition is seen predominantly in healthy young men without a history of previous lung disease. Twenty of the twenty-four patients in this study were males. The average age for this group of twenty-four patients was approximately twenty-eight years. The youngest patient was nineteen years, the oldest forty-six years.

2. The pathogenesis of this condition is not entirely clear. The present concept as to the origin of simple spontaneous pneumothorax is that it results from rupture of a valve vesicle or bleb situated on or near the pleural surface.

3. The clinical picture is usually one of sudden onset of pain on the side of the pneumothorax associated with shortness of breath. Both the symptoms and physical signs vary according to the degree of collapse and mediastinal displacement.

A history of physical exertion preceded the onset of spontaneous pneumothorax in only seven, or about 35 per cent, of the twenty-four patients presented in this study. In two instances, it followed the throwing of a baseball and snowball respectively and in three instances, spontaneous pneumothorax followed exertion while in an awkward position.

In the uncomplicated case of simple spontaneous pneumothorax in this study, the acute symptoms usually subsided in a few days and the time for complete re-expansion of the affected lung varied from one to eight weeks. The average period of time for complete re-expansion of the collapsed lung was five weeks.

4. It is estimated that recurrent spontaneous pneumothorax occurs in about 10 to 20 per cent of cases. Nine, or approximately 27 per cent, of the twenty-four patients studied experienced a recurrent pneumothorax. In seven instances the collapse involved the same side, while the remaining two patients had an alternating spontaneous pneumothorax.

5. The present day treatment of recurrent spontaneous pneumothorax is either by the production of a chemical pleuritis or by surgical intervention. Each method has its advocates. Two patients in this study have been treated by the induction of a chemical pleuritis and one patient by thoracotomy. All three patients have remained asymptomatic for periods varying from two to six years.

6. Twenty of the twenty-four patients reported in this paper have had adequate follow-up studies for periods varying from one to twelve years. Twenty of these twenty-four patients are in good health. One patient had a coronary occlusion recently and is still alive. The remaining three patients have not been adequately followed.

7. The prognosis in the uncomplicated case of simple spontaneous pneumothorax is excellent and recovery complete.

8. The conclusion reached from this case study agrees with that of other writers, namely that in subsequent years, active pulmonary tuberculosis develops in these patients no more frequently than in any other similar age group.

The author is indebted to Drs. H. D. Kerr, Lyell C. Kinney and Carl L. Gillies for their encouragement and valuable suggestions in the preparation of this paper.

1831 Fourth Ave.
San Diego 1, Calif.

REFERENCES

1. BLACKFORD, S. D. Spontaneous pneumothorax in college students. *J.A.M.A.*, 1939, *113*, 737–739.
2. BRUNNER, A. Zur Pathogenese und Therapie des Spontanpneumothorax. *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1921, *33*, 124.
3. FISHER, B. Der gutartige Spontanpneumothorax durch Ruptur von spitzennarbenblasenein typisches Krankheitsbild. *Ztschr. f. klin. Med.*, 1922, *95*, 1.
4. HARTZELL, H. C. Spontaneous hemopneumothorax; report of three cases and review of literature. *Ann. Int. Med.*, 1942, *17*, 496–510.
5. HAYASKI, J. Ueber totlichen Pneumothorax durch; Infarct und Emphysema. *Frankfurt. Ztschr. f. Path.*, 1915, *16*, 1.
6. HENNELL, H., and STEINBERG, M. F. Tense pneumothorax; treatment of chronic and re-

current forms by induction of chemical pleuritis. *Arch. Int. Med.*, 1939, *63*, 648–663.

7. Kirshner, J. J. Spontaneous pneumothorax; aetiological considerations. *Am. Rev. Tuberc.*, 1939, *40*, 477–481.
8. Kjaergaard, H. Spontaneous pneumothorax in the apparently healthy. *Acta med. Scandinav.*, (supp. 43) 1932, pp. 1–159; 1–93.
9. Kjaergaard, H. Pneumothorax simplex. Two cases with autopsy findings. *Acta med. Scandinav.*, 1933, *80*, 93–104.

9a. Leach, J. E. Pneumothorax in young adult males. *Arch. Int. Med.*, 1945, *76*, 264–268.

10. Leggett, E. A., Myers, J. A., and Levine, I. Spontaneous pneumothorax; report of 31 cases. *Am. Rev. Tuberc.*, 1934, *29*, 348–361.
11. Lorge, H. J. Etiology of iodiopathic pneumothorax. *Am. J. M. Sc.*, 1940, *199*, 635–641.
12. Ornstein, G. G., and Lercher, L. Spontaneous pneumothorax in apparently healthy individuals. *Quart. Bull., Sea View Hosp.*, 1942, *7*, 149–187.
13. Pease, P. P., Steuer, L. G., and Chapman, A. Spontaneous pneumothorax in soldiers. *Bull. U. S. Army Med. Dept.*, 1944, No. 82, 102–107.
14. Perry, K. M. A. On spontaneous pneumothorax, *Quart. J. Med.*, 1939, *8*, 1–21.
15. Pitt, G. N. Case of rapidly fatal haemopneumothorax apparently due to the rupture of an emphysematous bulla. *Tr. Clin. Soc. Lond.*, 1900, *33*, 95.
16. Ranking, W. H. Pneumothorax without urgent symptoms, followed by recovery: subsequent death of the patient from dissecting aneurism of the aorta. *Brit. M. J.*, 1860, p. 665.
17. Richmond, P. Spontaneous pneumothorax in the apparently healthy. *U. S. Nav. Med. Bull.*, 1940, *38*, 306–308.
18. Tyson, M. D., and Crandall, W. B. Surgical treatment of recurrent idiopathic spontaneous pneumothorax. *J. Thoracic Surg.*, 1941, *10*, 566–571.
19. Van Ordstrand, H. S. Idiopathic spontaneous pneumothorax. *Cleveland Clin. Quart.*, 1940, *7*, 178-183.
20. Waring, J. J. Spontaneous pneumothorax. National Research Council, Division of Medical Sciences, Issued by the Office of Medical Information, Washington (under grant of the Johnson & Johnson Research Foundation). 1944.
21. Wilson, J. L. Spontaneous pneumothorax. *Internat. Clin.*, 1937, *1*, 157–175.

BOECK'S SARCOID (SARCOIDOSIS)*

By ABRAHAM OPPENHEIM, M.D.,† *and* ROBERT S. POLLACK, M.D.†
NEW YORK, NEW YORK

BOECK'S sarcoid is a generalized systemic disease with protean manifestations. The disease is characterized by chronic duration, variable clinical picture and a usually benign course. In its tendency to affect many organs, it simulates the lymphomatous diseases.

A definite diagnosis can be established by biopsy. Since peripheral lymph nodes or lesions are not constantly present, biopsy is not always feasible. It seems, therefore, that a correlation should be established between the roentgenographic findings of the chest particularly, and an anatomic diagnosis of sarcoidosis.

The roentgenographic findings, especially of the chest, have been reported by many authors.[2,14,18] It seems unnecessary to classify the variants described into artificial subdivisions from what is usually a fairly characteristic and ofttimes diagnostic picture. The wide dissemination, the multiplicity of types of pulmonary shadows, and particularly the concomitant, symmetrical bilateral hilar enlargement are features which should lead one to suspect Boeck's sarcoid.[18] A distinct help in the diagnosis of pulmonary sarcoid is the spectacular discrepancy between the massive anatomical involvement and the paucity or absence of respiratory symptoms and physical signs.[14,18,19] Years ago Hunter[12] posed the question: "Are apparently healthy patients who give evidence of increased hilus shadows accompanied by diffuse infiltration of the lungs at x-ray examination . . . suffering from Hutchinson-Boeck's sarcoid?" We think they are.

In regard to the chest lesions, the unmistakable tendency is toward clearing, but the time required varies and may be long. Recent authors[5,18,20] have mentioned the lack of specific treatment. Nevertheless, there are others, mainly radiologists,[1,6,18] who have reported good results with radiation therapy. In our series of 42 cases, 24 were treated with radiation therapy and this method, although not conclusive, seems to represent a form of therapy beneficial to the patient.

The integration of the roentgenographic findings, clinical data, course and response to treatment should suggest a diagnosis of Boeck's sarcoid whenever recourse to biopsy is not possible. In this way numerous cases which heretofore have been relegated to the undiagnosed files may prove to be Boeck's sarcoid.

HISTORICAL DATA

In 1869 Jonathan Hutchinson[12] observed a patient with peculiar purplish raised lesions on legs, fingers and forearms. These cutaneous manifestations have since been associated with sarcoidosis and represent but one phase of the disease. Since then, the concept of the disease has undergone considerable change.

In succeeding years, Besnier,[3] Boeck,[4] Heerfordt,[22] Kuznitsky and Bittorf[16] added further material. In 1914 Schaumann[21] correlated these apparently unrelated clinical entities. Further progress was made when Jüngling[13] in 1919 described the roentgenographic findings in the metacarpal and tarsal bones.

Recently the disease has interested the internist, radiologist, surgeon and opthalmologist. Numerous reports have appeared in the literature under a variety of names: multiple benign sarcoid, benign miliary lupoid (Boeck), lupus pernio (Besnier), benign lymphogranulomatosis (Schaumann), benign caseating tuberculosis (Pinner), Hutchinson-Boeck's disease, Besnier-Boeck-Schaumann disease and others. The nomen-

* From the Medical Service of Dr. Lloyd F. Craver, Memorial Hospital for the Treatment of Cancer and Allied Diseases, New York, N. Y.
† Trainee, National Cancer Institute, Washington, D. C.

clature therefore has become quite unwieldy. In spite of the confused historical background the disease is most widely known as Boeck's sarcoid, and perhaps it would be best for the sake of clarity to retain this nomenclature.

SOURCE OF MATERIAL

The lymphoma register at Memorial Hospital is probably one of the largest in the country. Ample opportunity is thus afforded to observe the numerous types of cases of lymphadenopathy with or without chest involvement. However, it was not until 1939 that the difference between what is now called Boeck's sarcoid and the lymphomas was appreciated. Undoubtedly many cases of sarcoidosis were seen, but it was thought the chest roentgenograms represented a juvenile type of tuberculosis in an adult. The biopsy material was reported by Dr. James Ewing as hyperplastic tuberculosis. Nevertheless, these cases clinically resembled Hodgkin's disease.

The complicating factor of a negative tuberculin test vitiated a diagnosis of tuberculosis, as it was felt that the latter would usually give a positive result. Because of these factors, and the favorable clinical course, many cases fell into a group which was called atypical Hodgkin's disease, since they were neither true lymphomas nor tuberculosis. However, about 1939, the realization that Boeck's sarcoid was a clinical entity rather than an atypical variant became firmly established.

We have collected 42 cases from 1935 to the present. In our series we reviewed those cases proved by original biopsy; those which have been called hyperplastic tuberculosis, but which, on review of slide, are now classified as Boeck's sarcoid; and a group whose roentgenograms, clinical data, course and response to therapy seem to meet fully the requirements of Boeck's sarcoid. Included in our group are some cases showing involvement other than chest, such as skin, peripheral nodes, submaxillary gland and stomach.

Attempts were made to rule out tuberculosis in every case. Sputum examination, gastric washings and guinea pig inoculations were done whenever necessary. The hematological studies were confined to sternal aspirations and complete blood counts. The relationship of hyperproteinemia to Boeck's sarcoid was not investigated. During the past two years we have maintained the final tuberculin dilution reading at 1:100. Prior to that, no definite dilution factor was mentioned, the reading merely being recorded as positive or negative. This, of course, may serve as a source of error.

Roentgenograms of the hands were taken on admission only. It was felt that this represented a characteristic cross section of the group studied, as the patients were probably in various stages of the disease. Where definite peripheral adenopathy was present, biopsy was always obtained.

Therapy in the form of roentgen radiation was given to those patients who showed symptoms. All cases were closely followed: the longest for ten years, the shortest for two months. Two of our patients died: one following a radical neck dissection for papillary adenocarcinoma of the thyroid; the second died six years after initial admission. The cause of death as described by the patient's local doctor seemed to resemble a massive tuberculous involvement.

DISCUSSION

Our youngest patient was eleven and our oldest sixty-eight (Table 1). Five of our

TABLE I

Age	11 to 68 years—Average 27.5 years
Sex	19 male—23 female
Color	30 white—12 negro
Positive chest roentgenograms plus peripheral manifestations	20
Positive chest roentgenograms only	14
Peripheral manifestations alone	8
Proved by biopsy	27
Bone changes	1
Positive tuberculin test	13 or 36 per cent
Negative tuberculin test	23 or 64 per cent
Tuberculin test not done	6
Number treated with roentgen rays	24

FIG. 1. Shows extensive mediastinal and hilar involvement. Patient had shortness of breath.

patients ranged between forty-nine and sixty-eight. The major portion fell between twenty and thirty-five, with an average for the entire series of 27.5 years. This is in conformity with the general opinion that sarcoidosis occurs in the younger age groups.

There were 30 white and 12 negro patients in the present series, making an incidence of 28.5 per cent negroes. Other series have shown a proportion of negroes ranging from 76.6[17] to 85.7[19] per cent. To prove that sarcoidosis occurs more often in negroes than in whites, we must compare the proportion of negroes in the present series with the proportion of negroes in the population from which the series was drawn. In New York City, negroes form 6 per cent of the population. The percentage of negroes in our hospital registration is much less than that. Is the difference, therefore, between 28.5 per cent and 6 per cent a significant difference or could it have arisen by chance? An application of the statistical formula for the standard error of a percentage shows that this difference is significant and would occur by chance only about three times in one thousand trials. Therefore, we infer that negroes, in general, are more liable to sarcoidosis than white persons.

The presence of bone changes in sarcoidosis has been repeatedly stressed. Nevertheless, only one of our cases showed any change. Whether this was accidental or our patients were all in the same phase of the disease when first seen is difficult to state The latter is probably unlikely. Bone changes, therefore, are confirmatory signs only, and their absence should not preclude the diagnosis of Boeck's sarcoid.

The occurrence of a negative tuberculin test is still an important feature. Reisner[20] and Kissmeyer[15] both found 40 per cent positive; our series showed 36 per cent positive in those tested. There seemed to be no correlation as to age, sex, color or anatomical involvement. To find in a hospital population which is distinctly urban a negative tuberculin test in 64 per cent of a group appears to be of diagnostic importance.

No eosinophilia or unusual abnormality in the differential smear or sternal aspiration was noted in any of our cases.

FIG. 2. Minimal hilar and mediastinal involvement. Patient had no symptoms. Positive biopsy.

Involvement of many or all structures of the eye is comparatively common during some stage of the disease. One of our patients was totally blind, the uveitis present probably being on a sarcoid basis.

Because of the special nature of our institution, the major portion of our series consisted of those patients referred to us with mediastinal involvement of obscure nature. The possibility of a lymphomatous disease was always to be ruled out. Therefore, by far the largest group in our series, 34, showed chest manifestations. Twenty of these patients showed chest and peripheral manifestations, and 14 showed chest signs only.

Twenty-four of the 34 with chest involvement showed one or more of the following symptoms: dyspnea, shortness of breath, malaise, loss of weight, cough, chest pain and fever. Cough was the most prominent symptom. Weight loss and dyspnea were the least common. Nevertheless, the symptoms were out of all proportion to the large amount of chest involvement.

Figure 1, A.B., shows the roentgenogram of an eighteen year old negro male, whose only symptom was shortness of breath of seven months' duration. A routine chest roentgenogram taken at an Army Induction Center revealed a mediastinal mass. Physical examination also revealed peripheral lymphadenopathy. An excised lymph node from the right supraclavicular region was reported in our laboratory as Boeck's sarcoid.

Figure 2, M. S., shows the roentgenogram of a twenty-nine year old white male with no symptoms, but whose routine chest roentgenogram taken at an Army Induction Center revealed slight hilar adenopathy and mediastinal involvement. One small neck node was biopsied and was positive for Boeck's sarcoid.

Figure 3, J. B., illustrates the chest roentgenograms of a thirty-three year old female whose only complaint was weakness. The bilateral, symmetrical hilar adenopathy suggested Boeck's sarcoid despite a positive tuberculin test and the absence of metacarpal cysts. Examination revealed two very small lymph nodes (4 mm. across) in the right supraclavicular area and several shotty nodes of equal size in each axilla. All the nodes felt insignificant clinically. Despite their small size, the two palpable neck nodes were excised for biopsy. The pathological report was Boeck's sarcoid, a finding which would substantiate the impression that even the smallest palpable nodes in sarcoidosis may give histopathological evidence of the disease.

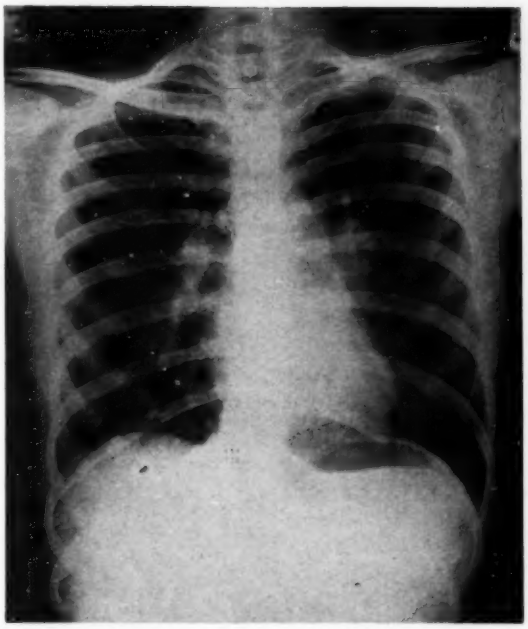


Fig. 3. Bilateral symmetrical hilar involvement. Patient's only complaint was weakness.

Figures 4 and 5, F. P., are those of a thirty-six year old white female whose history and physical signs are somewhat unusual. Illness began with chills and high fever, accompanied by a painful swelling of the left parotid gland. She was treated symptomatically at another institution where the chills and fever subsided in about one week. The parotid swelling, however, did not regress and she was referred to Memorial Hospital. Examination revealed bilaterally enlarged parotid glands, greater on the left. Sialograms of the left parotid gland showed a diffuse enlargement with a feathery appearance of the smaller ducts

FIG. 4. Left parotid enlargement accompanied by chills and fever. Aspiration biopsy of gland was positive for Boeck's sarcoid.

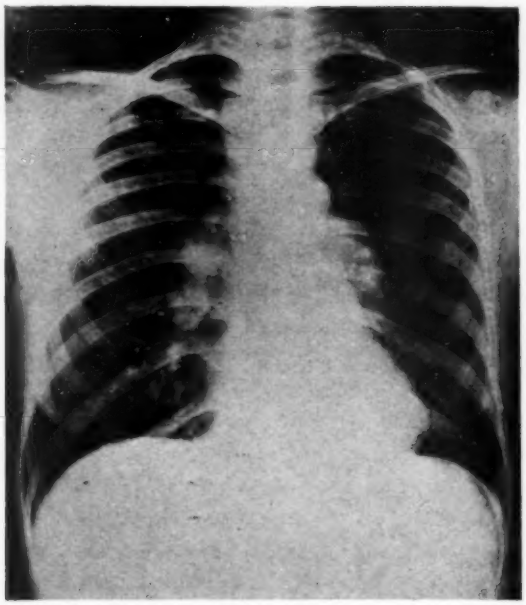


FIG. 5. Same patient as in Figure 4. Chest roentgenogram showing hilar and mediastinal involvement.

suggesting some generalized infiltrative process in the gland similar to Mikulicz's disease or chronic interstitial parotitis. A roentgenogram of the chest revealed large nodular shadows in both hila with widening of the upper mediastinum, particularly on the right. Aspiration biopsy of the left parotid gland was reported in our labora-

TABLE II

	Average Age	Color	Sex	Peripheral Manifestations	Splenic Enlargement	Tuberculin Reaction	Sternal Marrow Examination
Boeck's sarcoid	20–35 yr. 27.5 yr. in our series	More common in negroes	Equal	May be present	Rare	Usually negative. 64% in our series	Negative
Chronic lymphatic leukemia	Above 40 yr.	Predominantly white	Predominantly male	Usually present	Frequent	Unrelated. Percentage as in general population	Usually involved
Lymphosarcoma	47 yr. (all ages)	Predominantly white	Predominantly male, 7:3 ratio[24]	Usually present	Not infrequent. 21% palpable[24]	Unrelated. Percentage as in general population	Infrequently involved
Hodgkin's disease	20–30 yr.[5] 35 yr. mean[25]	Predominantly white	62% male[22]. Majority male	May be present	Not infrequent. 60% secondary involvement[5]	Steiner[23] stated, in 1934, that 71.5% were negative	Infrequent to no involvement

tory as Boeck's sarcoid. The tuberculin test was negative. Roentgenogram of the hands revealed no cystic changes. A mild iritis was present.

A very interesting case is that of J. R.,* a fifty year old white female, whose original complaint when first seen was the presence of a large mass in the left groin and swelling of the leg since October, 1934. The mass was removed surgically at another institution in 1935. The pathological report was "tumor, type unknown." In 1937 an epitrochlear node was excised at Memorial Hospital and was called giant follicular hyperplasia. In 1940, a node from the right groin was excised and reported as Boeck's sarcoid or tuberculosis. About this time she developed epigastric discomfort. A gastrointestinal series revealed a small ulcer on the lesser curvature which looked like carcinoma. The patient had a partial gastrectomy in 1941 along with a liver biopsy. The pathological report of both tissues was Boeck's sarcoid. Roentgenograms of the chest and small bones of the hands were negative. No tuberculin test or sputum examinations were done. The patient did well and showed no evidence of disease on subsequent visits, until May, 1945. At this time she was observed to have a group of enlarged, firm lymph nodes in the right groin. These were excised and reported to be Hodgkin's disease. All of the previous slides were again reviewed with no change in the original interpretation of each.

This patient presented four distinct clinical entities during her course of illness. It is probable, however, that all of these conditions are somewhat related. In this case it appears that Boeck's sarcoid was an intermediate phase between tuberculosis and lymphoma. It has been our clinical impression that sarcoidosis may at some time eventuate into one of these conditions. Do these entities represent different levels of allergic response?

Reisner[20] states that there is reason to believe that tuberculosis is not merely an incidental complication but that its potentiality of development is inherent in sarcoidosis. While we have no evidence to substantiate that claim, and while the relationship of sarcoidosis to tuberculosis is still a moot point, biopsy study of the nodes of one of our patients showed a transition

* Case J. R. is from the Gastric Service of Dr. George T. Pack.

Roentgenogram of Chest	Bone Involvement	Response to Radiation Therapy	Course	Prognosis
Usually shows bilateral hilar adenopathy. Occasional mediastinal widening and parenchymal infiltration	May be present as cysts, especially in metacarpal and metatarsal bones. Usually painless	Sensitive to a small dose; rare recurrences	Chronic, benign; regresses even without treatment. May be asymptomatic	Good. Rarely fatal. Relationship to tuberculosis disputed
11% positive[7]. Usually negative; may show mediastinal widening	7% involved[9]. Usually painful; majority osteoporotic. Many sites	Radiosensitive; frequent recurrences	Chronic, increased severity. Ultimately fatal	3–5 years average duration of life. 36–42 months mean survival rate. 15% five year survival
12% positive[24]. 39% autopsy positive[7]. Usually negative. May show mediastinal widening	10.4% involved[3]. Usually painful; majority osteolytic. Many sites	Presumably sensitive. Occasionally resistant. Frequent relapses	Slowly progressive with variable remissions, but always fatal	16% five year survival
Usually shows mediastinal widening and/or hilar adenopathy. 34% roentgen-ray positive, 64% autopsy positive[7]	15% involved[25]. Usually painful; majority osteoplastic and osteolytic. Many sites	Usually responds; frequent recurrences	Frequent remissions and relapses. Ultimately fatal	30–32 months mean survival rate. 18% five year survival

between typical tubercle and sarcoid. The patient's tuberculin test was positive. Acid fast stains of the node material were negative. One month later another node in the same area showed caseous tuberculosis on section.

DIFFERENTIAL DIAGNOSIS

The clinician has encountered great difficulty in accurately differentiating sarcoidosis from the lymphomas, especially where there are no outstanding signs except the roentgenographic findings. It is in these cases that the clinical features of the disease assume far greater significance. Hodgkin's disease, because of a closely simulating chest picture and other clinical features, is the chief condition from which a differential diagnosis must be made. Nevertheless, any of the lymphomas due to their bizarre nature can simulate sarcoidosis. This was illustrated by a recent case in which the chest roentgenograms and symptoms suggested Boeck's sarcoid but a lymph node biopsy proved to be Hodgkin's disease. In the accompanying table (Table II), we have listed the main differential points.

TREATMENT

It has been our practice to use roentgen therapy only in those cases manifesting symptoms. Other forms of therapy such as ultraviolet radiation, arsenicals, iodides, etc., are merely symptomatic forms of treatment and do not affect the disease directly.

In cases where symptoms are present, definite benefit is derived from the administration of small doses of roentgen radiation to the mediastinum and to peripheral nodes if the latter are palpably enlarged. Where surgical intervention will eliminate the disease in toto it is to be preferred. This latter form of treatment is exemplified in the case of sarcoidosis of the stomach.

We have treated 24 patients in the past ten years. Twenty have been observed sufficiently long to justify accurate conclusions. Three showed no decrease in the amount of disease in the region irradiated. The appearance of parenchymal infiltration was subsequently noted. Despite the increase in the lung involvement and the stationary phase of the mediastinal mass following radiation therapy, there was no accentuation of the clinical symptoms. These cases will receive a second course of therapy.

Our method of treatment has been to give six exposures of 100 r (in air) to the anterior and posterior mediastinum (fields vary depending on the size of the mass to be treated), one field daily, using 250 kv., 1.5 mm. Cu, 70 cm. skin target distance. Peripheral nodes are treated at 50 cm. skin target distance. The total dose varies from 100 to 400 r at one exposure, the other factors remaining the same. Following treatment an interval of about two months is allowed to elapse for observation. At the end of this time if disease is still present, another course is usually given.

PROGNOSIS AND CURE

Our series seems to indicate that the prognosis of Boeck's sarcoid is favorable. The duration and chronicity of the disease make long-standing follow-up necessary. We have noted regression and complete disappearance of the mediastinal and peripheral lesions with radiation therapy. Symptoms when present have usually abated. While it is true that spontaneous regression with apparent recovery may take place in the absence of treatment, we have as yet no diagnostic criterion enabling us to anticipate which patients will fall into that group. Since irreparable damage can often accompany progressive organic involvement, cautious radiation therapy may offer a method of allaying future damage and may thus make a favorable prognosis more likely.

SUMMARY

1. A study of 42 cases of Boeck's sarcoid is presented.

2. The majority of the cases had mediastinal and peripheral node involvement.

3. A differential diagnosis between

Boeck's sarcoid and the lymphomas is presented.

4. Radiation therapy of Boeck's sarcoid is discussed.

CONCLUSION

Radiation therapy is indicated in Boeck's sarcoid when symptoms are present.

Memorial Hospital
444 East 68th St.
New York 21, N. Y.

REFERENCES

1. Bernstein, S. S., and Oppenheimer, B. S. Boeck's sarcoid; report of 6 cases with one necropsy. *J. Mt. Sinai Hosp.*, 1942, *9*, 329–343.
2. Bernstein, S. S., and Sussman, M. L. Thoracic manifestations of sarcoidosis. *Radiology*, 1945, *44*, 37–43.
3. Besnier, E. Lupus pernio de la face; synovites fungueuses (scrofulotuberculeuses) symétriques des extrémités supérieures. *Ann. de dermat. et syph.*, 1889, *10*, 333–336.
4. Boeck, C. Multiple benign sarcoid of the skin. *J. Cutan. & Genito-Urin. Dis.*, 1899, *17*, 543–550. Fortgesetzte Untersuchungen über das multiple benignes Sarcoid. *Arch. f. Dermat. u. Syph.*, 1905, *73*, 71–86; 301–332.
5. Cecil, R. Medicine. Sixth edition.
6. Craver, L. F. Personal communication to authors.
7. Craver, L. F., Braund, R. R., and Tyler, H. Y. Lesions of lungs in lymphomatoid diseases. Am. J. Roentgenol. & Rad. Therapy, 1941, *45*, 342–349.
8. Craver, L. F., and Copeland, M. M. Lymphosarcoma in bone. *Arch. Surg.*, 1934, *28*, 809–824.
9. Craver, L. F., and Copeland, M. M. Changes of bones in the leukemias. *Arch. Surg.*, 1935, *30*, 639–646.
10. Harrell, G. T. Generalized sarcoidosis of Boeck. *Arch. Int. Med.*, 1940, *65*, 1003–1034.
11. Heerfordt, C. F. Ueber eine febris Uveoparotidea subchronica an der glandula Parotis und der Uvea des Auges lokalisiert und haufig mit paresen cerebrospinalen Nerven kompliciert. *Arch. f. Ophth.*, 1909, *70*, 254.
12. Hunter, F. T. Hutchinson-Boeck's disease (generalized "sarcoidosis"). *New England J. Med.*, 1936, *214*, 346–352.
13. Jüngling, O. Ostitis tuberculosa multiplex cystica (eine eigenartige Form der Knochentuberculose). *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1919–1921, *27*, 375–383.
14. King, D. S. Sarcoid disease as revealed in chest roentgenogram. Am. J. Roentgenol. & Rad. Therapy, 1941, *45*, 505–512.
15. Kissmeyer, A. La maladie de Boeck: Sarcoïdes cutanées bénignes multiples. Levin & Munksgaard, Copenhagen, 1932.
16. Kuznitsky, E., and Bittorf, A. Boecksches Sarkoid mit Beteiligung innerer Organe. *München. med. Wchnschr.*, 1915, *62*, 1349.
17. Longcope, W. T. Sarcoidosis, or Besnier-Boeck-Schaumann disease: Frank Billings lecture. *J.A.M.A.*, 1941, *117*, 1321–1327.
18. Pohle, E. A., Paul, L. W., and Clark, E. A. Roentgen therapy of Boeck's sarcoid. *Am. J. M. Sc.*, 1945, *209*, 503–513.
19. Reisner, D. Boeck's sarcoid and systemic sarcoidosis (Besnier-Boeck-Schaumann disease). *Am. Rev. Tuberc.*, 1944, *49*, 289.
20. Reisner, D. Boeck's sarcoid and systemic sarcoidosis (Besnier-Boeck-Schaumann disease). *Am. Rev. Tuberc.*, 1944, *49*, 437–462.
21. Schaumann, J. Sur le lupus pernio. Memoire presente en Novembre, 1914, a la Société Française de Dermatologie et de Syphiligraphie pour le Prix Zambaco.
22. Slaughter, D. P., and Craver, L. F. Hodgkin's disease. Am. J. Roentgenol. &. Rad. Therapy, 1942, *47*, 596–606.
23. Steiner, P. E. Etiology of Hodgkin's disease; skin reaction to avian and human tuberculin proteins in Hodgkin's disease. *Arch. Int. Med.*, 1934, *54*, 11–17.
24. Sugarbaker, E. D., and Craver, L. F. Lymphosarcoma. *J.A.M.A.*, 1940, *115*, 17–23; 112–117.
25. Vieta, J. O., Friedell, H. L., and Craver, L. F. Survey of Hodgkin's disease and lymphosarcoma in bone. *Radiology*, 1942, *39*, 1–15.

PATTERN TYPES IN THE SMALL INTESTINE*

By SYDNEY M. FRIEDMAN, M.D., PH.D.
MONTREAL, QUEBEC

ALTERATIONS in the roentgenologic pattern of the small intestine have recently received deserved recognition in the literature. Since 1933 when Mackie[9] described pattern changes associated with non-tropical sprue, the field has been much extended. It was shown that similar derangement may occur in chronic idiopathic steatorrhea in adults,[15] acholia,[8] diarrhea,[5] tetany,[12] and nutritional deficiencies.[6,7,10,11] It would seem that these changes are not necessarily characteristic of any one disease, but occur in many metabolic upsets in which the alimentary system is involved either primarily or secondarily. Ruffin, Baylin and Cayer[14] would classify the changes as mild, moderate or severe according to the criteria of coarsening of folds, segmentation, spasm and flocculation.

Based on anatomical findings[7] and clinical studies of the infant,[16] pattern derangements have been attributed to a common mechanism, probably the intramural nervous connections of the alimentary system, with or without disturbances of the mucosa. Apparently no attempts have been made to determine whether any factor of a more extrinsic nature is common to any of these clinical conditions which have been seen to produce changes. It has been pointed out that pattern changes of the small intestine may occur in apparently healthy subjects.[14] It is difficult to assess these findings since precise knowledge of the factors producing the pattern change is lacking and despite the amount of work carried on to investigate pattern changes, very little information has appeared concerning the normal pattern. Feldman,[4] feeling the need for this, described the roentgenologic appearance of the normal duodenum but did not state the number of subjects in his series nor his criterion of a normal subject. Pendergrass, *et al.*[13] have demonstrated the importance of the standard bland type of barium meal that must be used if other variables are not to be introduced. Chamberlin[3] described the appearance of the small intestine, but again stated no criteria of a normal subject. Those studies on children[16] have been on small series.

In view of this situation the following series is presented in an effort to establish the frequency both of normal and abnormal patterns.

For the present series roentgenograms taken in the Royal Victoria Hospital during the period 1942–1943 were selected. Only those patients whose subsequent hospital course revealed no detectable lesion of either the alimentary tract or parenteral viscera were retained for consideration.

Duodenum. In Table 1 the data for the series are presented. A normal appearance of the duodenum, according to the standard below, was noted in 95 of 110 male and 161 of 183 female subjects.

The normal pattern of the duodenum, although subject to fairly wide variation, shows certain definite characteristics in its roentgenographic features. In the first stage there are longitudinal folds during contraction which disappear as the cap fills. In that part of the second stage which is above the ampulla, there is, in most instances, an absence of folds. The ampulla itself is usually discernible as a small round negative shadow. Transverse folds, broken in the middle, appear at the ampulla or a little above and increase in size and continuity through the remainder of the second stage. Thickening of the folds and an increase in their number gives the third

* From the Department of Anatomy, McGill University, Montreal, Quebec. This work was carried on with the financial aid of the Blanche Hutchinson Fund of McGill University.

Fig. 1. Normal duodenum and small intestine. Note constriction in the middle of the third stage of duodenum which is discussed in text.

Fig. 2. The duodenal pattern is coarsened and the duodenum itself is uniformly large. This is classed as type 3 duodenum but shows some of the features of type 6. The intestine was hypermotile and the head of the meal was observed in the terminal ileum within one hour.

stage an appearance intermediate between that of the duodenum and the fine reticulated or feathery pattern of the jejunum.

The first variation encountered (type 1) was the presence of transverse folds in the first stage of the duodenum, noted in serial roentgenograms. This was seen in 2 females and 3 males.

The second variation (type 2) consisted of a duodenum dilated as far as the ampulla. This was not merely a large well filled cap, nor was it mere dilatation due to the presence of barium in process of passage. Notches of contraction were present in the adjacent wall, and in serial roentgenograms only small amounts of barium

Table I

DUODENAL PATTERN CLASSIFIED BY TYPES AND LISTED AS NUMBER OF CASES IN EACH AGE GROUP, MALE AND FEMALE

Age	0–10	11–20	21–30	31–40	41–50	51–60	61–80
	F M	F M	F M	F M	F M	F M	F M
Normal	2–2	11–6	42–13	29–31	36–18	27–15	14–10
Type 1		–1	1–		1–	– 2	
Type 2			1– 1	1–		1– 1	
Type 3			1–	2–	1– 2	– 1	
Type 4	–2		1–		1–		1–
Type 5	–1		– 1	1– 1	2–		
Type 6			2– 1	1–	1– 1	3–	

FIG. 3. Duodenum of a seven year old girl. Parents stated that the child had "vomited all her life." Roentgenoscopic examination was negative and a diagnosis of normal made. This pattern is classed as type 4 duodenum.

FIG. 4. Duodenum is classed as type 5 while remainder of small intestine is type 4. The appearance here suggests spasticity with irregular passage of barium.

were moved on. Even when no opaque medium was present the lumen remained large. This variation was noted in 3 female and 2 male subjects.

A duodenum whose lumen did not alter progressively in size, that is a continuously small or large lumen, was noted several times. The uniformly large lumen (type 3) occurred in 4 female and 3 male subjects. The uniformly small lumen (type 4) occurred in 3 female and 2 male subjects. It is

FIG. 5. Type 6 duodenum. This is probably a duodenitis.

worth noting that this latter type occurred in 2 of the 7 subjects under ten years of age.

A pattern in which the barium did not outline the folds but rather was disposed in globular masses (type 5) was noted in 3 female and 3 male subjects.

Course thick rugae giving a rococco or scroll-like pattern to the roentgenogram (type 6) were noted in 7 female and 2 male subjects in the present series. The appearance was suggestive of a duodenitis as described by Cordiner.[2]

The regular occurrence of a constricted

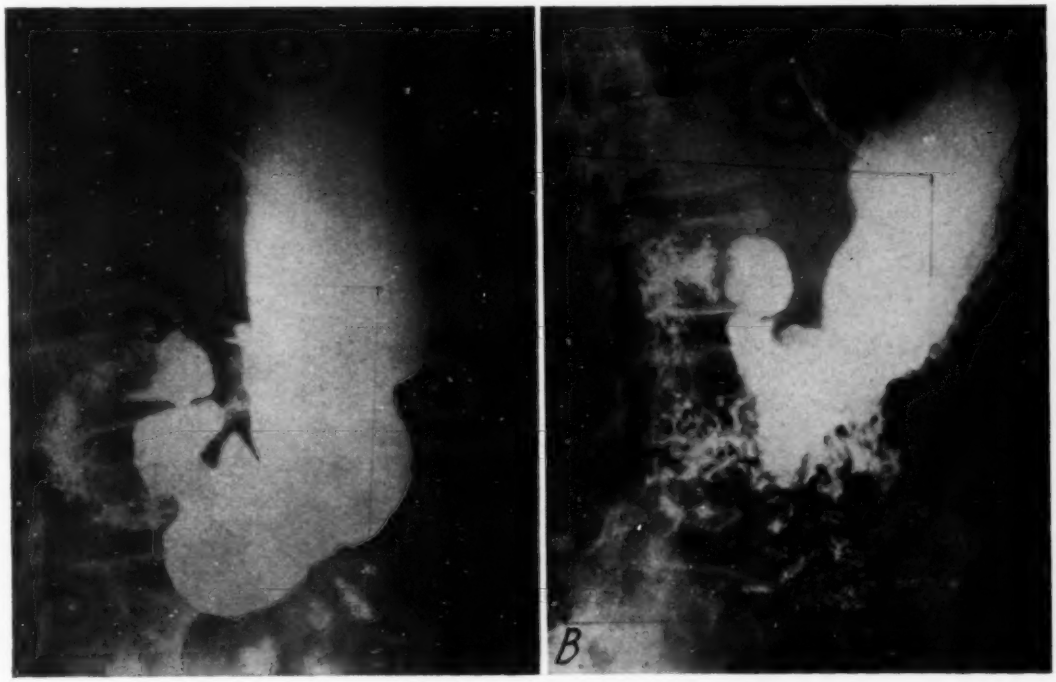


Fig. 6, *A* and *B*. Six years before, this man had pulmonary tuberculosis, at which time duodenum was normal. On basis of present roentgenoscopic examination diagnosis is "normal small intestine." Note marked alteration in shape of the duodenum and coarsening of the pattern.

appearance of the duodenum in the middle of the third stage has been described as due to a peristaltic wave.[4] This occurred 48 out of 293 times in the present series. It was so constant in appearance and position as to suggest a relation to the position of the superior mesenteric vessels.

A markedly swollen ampulla was noted in the serial roentgenograms of one patient.

Jejunum and Ileum. In the examination of the small intestine the normal pattern as defined by many authors[3,7,13,14] was noted in 61 of 78 subjects (Table II). Excessively thick rugae in the upper part of the small bowel (type 1) were noted in 4 subjects, while heavy irregular folds (type 2) also occurred in 4 subjects. Irregular passage of barium and a definite "snowflake" pattern (type 3) occurred in 7 subjects, while a spastic small bowel (type 4) much like that described by Barden, *et al.*[1] in the case of hypoproteinemia in dogs was noted in 2 male subjects.

DISCUSSION

While no attempt has been made to explain the definite pattern changes noted,

Table II

SMALL INTESTINE PATTERN CLASSIFIED BY TYPES AND LISTED AS NUMBER OF CASES IN EACH AGE GROUP, MALE AND FEMALE

Age		11–20	21–30	31–40	41–50	51–60	61–80
Normal	M	2	12	10	9	7	3
	F	0	2	6	5	5	
Type 1	M	0	1	0	1	1	0
	F	0	0	1	0	0	0
Type 2	M	0	1	0	0	1	1
	F	0	0	0	1	0	0
Type 3	M	0	3	0	1	1	0
	F	0	1	0	1	0	0
Type 4	M	1	0	0	0	1	0
	F	0	0	0	0	0	0

Fig. 7. Coarsening and irregularity of jejunal folds This pattern is classed as type 1 small bowel.

Fig. 8. Gastrojejunostomy. The jejunal pattern indicates coarsening of folds and irritability of this segment of small intestine. Patient's symptoms were suggestive of stomal ulcer which could not be found. This pattern is classed as type 1 small intestine.

several features are of interest. Although the patients in this series were discharged from the hospital as normal, it is possible that the gastrointestinal complaint for which they were examined had an undiagnosed organic basis in these pattern changes. On the other hand, it is possible that these changes may occur without any organic basis in normal subjects.

The present series shows that pattern variations of several definite types may occur. They are not specific for any one nutritional derangement or metabolic upset but only indicate a derangement common to many states and are perhaps even within the normal range.

CONCLUSIONS

Analysis of the serial roentgenograms of the small intestine of approximately 300 patients diagnosed as normal has been presented. It has been shown that six different pattern types in the duodenum and four definite types in the remainder of the small bowel may occasionally be distinguished.

I am indebted to Dr. Carleton B. Peirce, Professor of Radiology, McGill University, for his cooperation in the compilation of these data.

Department of Anatomy
McGill University
Montreal, P.Q.

REFERENCES

1. Barden, R. P., Thompson, W. D., Ravdin, I. S., and Frank, I. L. Influence of serum protein on motility of small intestine. *Surg., Gynec. & Obst.*, 1938, *66*, 819–821.
2. Cordiner, G. R. M. Duodenitis. *Brit. J. Radiol.*, 1942, *15*, 33–38.
3. Chamberlin, G. W. Roentgen anatomy of small intestine. *J.A.M.A.*, 1939, *113*, 1537–1541.
4. Feldman, M. Roentgenologic consideration of normal mucosa of the duodenum. *Radiology*, 1941, *37*, 284–288.
5. Golden, R. Small intestine and diarrhea. Am. J.

Roentgenol. & Rad. Therapy, 1936, *36*, 892–901.

6. Golden, R. The small intestine in vitamin B deficiency. *J.A.M.A.*, 1941, *117*, 913–917.
7. Golden, R. Abnormalities of small intestine in nutritional disturbances; some observations on their physiologic basis. *Radiology*, 1941, *36*, 262–286.
8. Gutzeit and Kuhlbaum. Ueber die Darmmotilität beim Ikterus. *München. med. Wchnschr.*, 1934, *81*, 1095–1098.
9. Mackie, T. T. Nontropical sprue. *M. Clin., North America*, 1933, *17*, 165–184.
10. Mackie, T. T. Vitamin deficiencies and the small intestine. *J.A.M.A.*, 1941, *117*, 910–912.
11. Mackie, T. T., and Pound, R. E. Changes in gastro-intestinal tract in deficiency states, with special reference to small intestine: roentgenologic and clinical study of 40 cases. *J.A.M.A.*, 1935, *104*, 613–618.
12. Pendergrass, E. P., and Comroe, B. I. Roentgen study of gastrointestinal tract in chronic idiopathic adult tetany. Am. J. Roentgenol. & Rad. Therapy, 1935, *33*, 647–656.
13. Pendergrass, E. P., Ravdin, I. S., Johnston, C. G., and Hodes, P. J. Studies of small intestine; effect of foods and various pathologic states on gastric emptying and small intestinal pattern. *Radiology*, 1936, *26*, 651–662.
14. Ruffin, J. M., Baylin, G. J., and Cayer, D. Clinical significance of alteration of the small intestine pattern as demonstrated by x-ray. *Gastroenterology*, 1945, *4*, 289–295.
15. Snell, A. M., and Camp, J. D. Chronic idiopathic steatorrhea; roentgenologic observations. *Arch. Int. Med.*, 1934, *53*, 615–629.
16. Zwerling, H., and Nelson, W. E. Roentgenologic pattern of the small intestine in infants and children. *Radiology*, 1943, *40*, 277–282.

PERITONEOPERICARDIAL DIAPHRAGMATIC HERNIA*

REPORT OF A CASE IN A NEWBORN INFANT SUCCESSFULLY CORRECTED BY SURGICAL OPERATION WITH RECOVERY OF THE PATIENT

By ANGUS K. WILSON, M.D., WILLIAM R. RUMEL, M.D.,
and O. L. ROSS, M.D.
SALT LAKE CITY, UTAH

INASMUCH as only 8 previous cases of this rare condition have been discovered in a careful search of the literature, since none of those described by others have survived operation for correction, and because it is felt that the condition can be recognized by proper evaluation of clinical and roentgenological findings and corrected by surgical operation, the following case is reported:

CASE REPORT

F. C., a white male infant was born in St. Mark's Hospital at 10:00 P.M. on November 21, 1945 after a normal left occipito-anterior delivery (John Cutler, M.D.), the second stage having lasted for about fifteen minutes. The patient's birth weight was 5 lb. 6 oz. and from the mother's menstrual history it was estimated that the birth was approximately two weeks premature. This was the mother's fourth pregnancy; the other three children were healthy and free from known congenital defects.

When cyanosis was noticed shortly after birth the infant was placed in an incubator and given oxygen by inhalation for twenty-four hours, at the end of which time his color was good. On the third neonatal day, cyanosis occurred again but it was very slight and the infant's color thereafter remained good until discharge from the hospital with his mother on November 30, 1945. The infant showed an initial weight loss of 6 oz., but he had regained his birth weight on discharge and was apparently healthy and well at that time. During the subsequent two weeks, his mother noticed nothing abnormal but on December 14, 1945, the infant "choked" when fed by breast and when given warm water. The choking spells became progressively worse with each feeding and on December 16, 1945, he became very cyanotic immediately after a feeding; the cyanosis cleared when he vomited. Following this episode several feedings were retained without development of cyanosis but from December 17, 1945, the infant choked and became cyanotic with each feeding. Relief of cyanosis was obtained by vomiting but after each attack he became very lethargic and would doze for several hours. Despite these attacks the bowel movements were four to five daily in number and of normal color. There was no evidence of cardiac tamponade (dilatation of veins of the neck, etc.) at any time.

On December 20, 1945, the cyanotic and vomiting episodes became more severe and the mother feared the infant might die. He was weaned and placed on bottle feedings but the formula was vomited and for the next nine days cyanosis and vomiting of varying intensity and duration persisted. Finally, on December 29, 1945, he became very cyanotic after a feeding and lost consciousness for about five minutes. One of us (O.L.R.) was called and the infant was immediately hospitalized and placed in an oxygen tent.

Weight on admission was 6 lb. 1 oz. Examination showed slight abdominal distention but no other abnormal findings except in the chest. Breath sounds were absent over the entire left chest except in the apex where there were a few scattered moist rales with bronchial type of breathing. The right lung was apparently not abnormal. The apex beat of the heart could not be felt and the cardiac sounds were very distant. Cardiac rate was 120–130 per minute. Because of the infant's condition, he was kept in the oxygen tent overnight with tentative working diagnosis of (1) congenital pulmonary atelectasis with right heart failure, (2) esophageal diverticulum, or (3) congenital anomaly of the gastrointestinal tract. By the following morning, he was much improved and it was considered proper to undertake investigative studies. Oxygen was used occasionally during the subsequent three days but cyanosis was not so severe during that time as upon admission. It was noted that when cyanosis developed it

* Read before the meeting of the Rocky Mountain Radiological Society, Denver, Colorado, August 8–10, 1946.

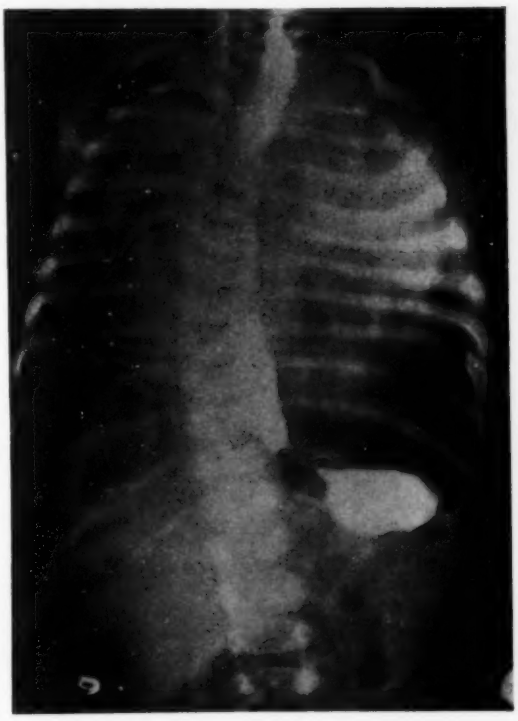


Fig. 1. Appearance December 30, 1945, showing normal esophagus and dense shadow obscuring upper lung field, especially on left side. The presence of mottled radiolucent areas, seen in the right upper half, with sharply defined delimitation, indicative of pericardial sac, should arouse suspicion of peritoneopericardial diaphragmatic hernia.

came on from half an hour to two hours after feedings.

On December 30, 1945, the day following admission he was referred for roentgen examination (A.K.W.) of the chest and esophagus (Fig. 1). There was complete opacity of the left lung field with the exception of a small illuminated patch in the costophrenic angle. The right side of the heart was much more prominent than usual but no pulsation of the shadow was seen roentgenoscopically. The right lung field appeared to be well aerated. The completeness of opacity on the left side suggested atelectasis of the upper lobe but a loculated shadow in the costophrenic angle and within the dense opacity

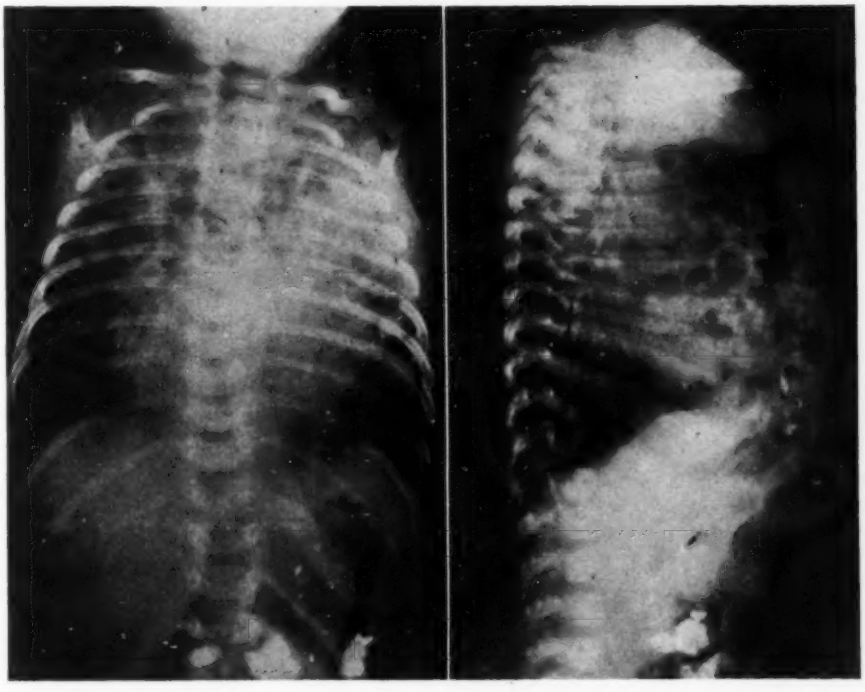


Fig. 2. January 3, 1946. Anteroposterior and lateral roentgenograms. Note the mottled loculated radiolucent shadows entering the thorax from below, anteriorly and slightly to the left. In the anteroposterior view these shadows are seen sharply delimited on the right side.

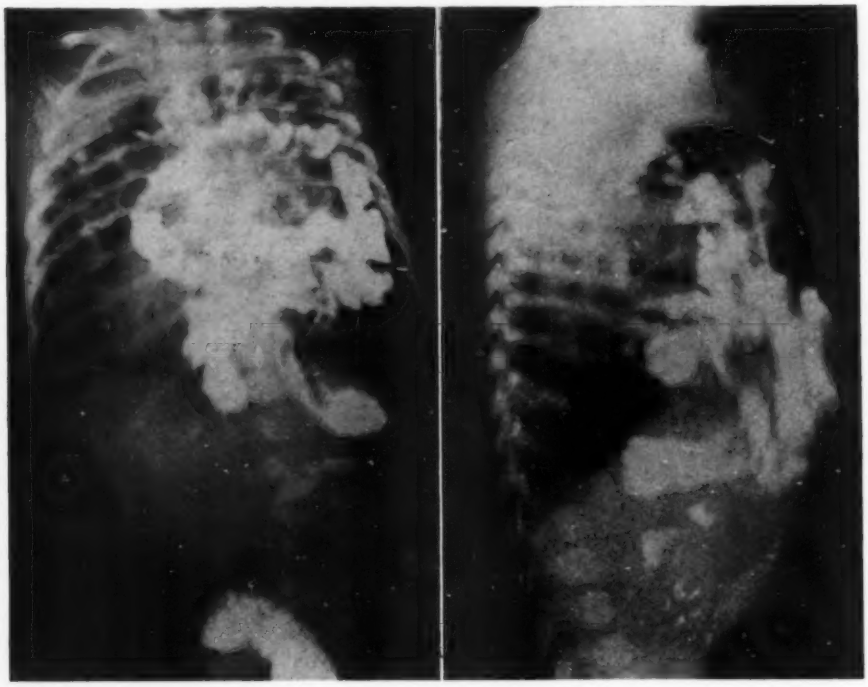


FIG. 3. January 4, 1946, following administration of two teaspoonfuls of barium sulphate powder in each of six feedings. The point of entry of intestines into the thorax is more clearly seen and the delimiting borders are more easily discernible. Some coils of intestine filled with gas are seen on the right and superiorly. The distal portion of the stomach enters the thorax through the hernia opening but at operation it was not found in the pericardium, apparently having reduced spontaneously.

presented the appearance of intestines in the thorax, indicating a possible diaphragmatic hernia, and it was recommended that additional studies after ingestion of barium sulphate suspension be undertaken when the patient's condition became suitable. A small amount of barium given at the time of this examination showed no abnormality of the esophagus.

Attempts at barium enema study of the colon were unsuccessful owing to the patient's inability to retain the enema. On January 2, 1946, he was given a teaspoonful of barium sulphate powder in each of six feedings. Examination the following morning, January 3, 1946, showed insufficient barium in the intestines to outline the bowel but centrally and in the region of density in the left upper chest there were a large number of loculated, gas-containing shadows interpreted as being coils of small intestine (Fig. 2). At this time (January 3, 1946) both the right and left borders of the loculated zone extended almost to the thoracic wall and were sharply defined. In view of subsequent findings, these were the margins of the pericardial sac. The only aerated portions of lung on either side were in the base. The lateral view showed the loculated shadows entering the thorax anteriorly. Re-examination the following day (Fig. 3) after additional barium feedings, double the amount previously given, showed barium-filled coils of intestine centrally and on the left side with some air-containing coils on the right. The intestine entered the thorax through an anterior defect almost in the midline. The cardiac shadow was obscured and no pulsation was seen on roentgenoscopy. On the basis of these findings, a diagnosis of diaphragmatic hernia was made with contents consisting of part of the stomach and the small intestine. The fairly sharp demarcation in the right hemithorax was thought to be due to the presence of a peritoneal sac about the hernia or shift of the mediastinum to the right. Herniation into the pericardium was not considered among the diagnostic possibilities because the existence of such a condition was not suspected.

After preliminary preparation, surgical exploration and correction were undertaken by

one of us (W.R.R.) on January 9, 1946, in an operation lasting two hours and ten minutes. Anesthesia (S. M. Smith, M.D.) was by D-tubo curarine and oxygen, a total of 18 U. being administered during the course of the operation. Surgical approach was through an incision over the left seventh rib from the anterior axillary line to the lateral margin of the right rectus muscle. The seventh rib was removed and the thoracic cavity was entered through an incision in its bed.

The left lung was found partially compressed but not airless. There was no herniation of viscera into the pleural cavity but the pericardial sac was distended and displaced to the left. Therefore an incision was made through the parietal pericardium anterior to the phrenic nerve, whereupon approximately three-fourths of the small intestine was found within the pericardium. A very thin layer of parietal peritoneum separated some coils of intestine from the parietal pericardium but it was evident that this layer was not continued upward since other coils were in direct contact with the heart. (Fig. 4).

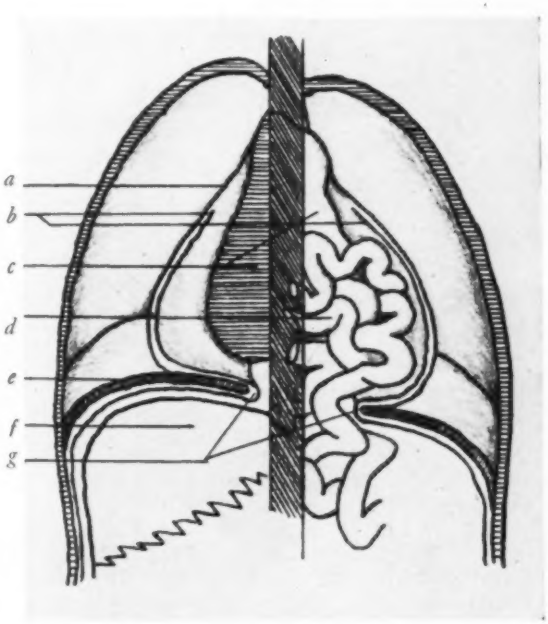


Fig. 4. Diagrammatic sketch (W.R.R.) of appearance at operation after opening pericardial sac. (*a*) pericardium; (*b*) hernial sac. Attachment of upper reflections was not determined; (*c*) heart; (*d*) small intestine overlying heart; (*e*) diaphragm; (*f*) liver; (*g*) margins of hernia opening.

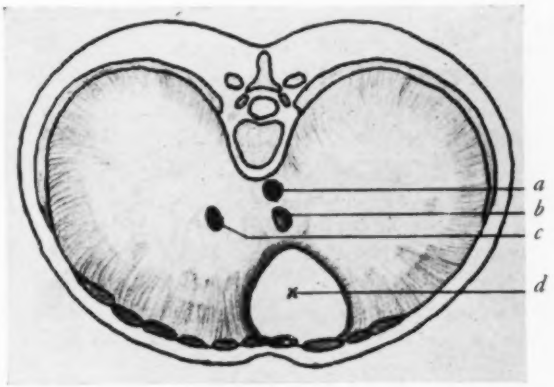


Fig. 5. Diagrammatic sketch (W.R.R.) showing relations of defect after reduction of herniated intestine. (*a*) aortic opening; (*b*) esophageal opening; (*c*) vena cava opening; (*d*) pericardioperitoneal diaphragmatic defect.

After reduction of the herniated intestine, there was found a triangular defect in the diaphragm (Fig. 5) having its base anteriorly and its apex just in front of the esophageal hiatus. The right margin originated just to the right of the sternum and the left margin at the parasternal line. The margins of the defect were thickened and firm at all points. There was a definite muscular layer immediately anterior to the esophageal hiatus. The cardiac portion of the stomach was beneath the diaphragm. The left lobe of the liver was seen through the defect and was irregular, small, and congenitally deformed. Other organs immediately adjacent to the orifice were not abnormal, but in order to avoid undue trauma, careful examination of all abdominal contents was not made.

The margins of the defect were isolated and using interrupted sutures of No. 20 cotton, the posterior two-thirds of the defect was repaired. The remaining defect was obliterated by suturing the margins to the anterior thoracic wall. The entire suture line was then reinforced with additional mattress sutures of No. 40 cotton. A counter incision was made in the pericardium about 2 cm. posterior to the phrenic nerve to prevent a pericardial effusion under tension and the anterior pericardial incision was closed loosely with interrupted sutures after thorough irrigation of the pericardial and pleural cavities with warm physiologic saline solution. Penicillin, 50,000 units, was injected into the pericardium and the pleura. The incision through the rib bed was ap-

proximated with continuous No. 000 chromic catgut. The remaining wound was closed in layers with interrupted sutures of No. 60 cotton. Before final closure, the lung was inflated with positive pressure and air was aspirated from the pleural space through a previously placed catheter. The catheter was then withdrawn.

The immediate postoperative condition was good and the postoperative course essentially uneventful, the only complication being the development of a slight head cold. For this and also for prophylaxis the patient received penicillin in intravenous Ringer's solution.

Roentgen examination (A.K.W.) January 26, 1946, three weeks after operation, by roentgenoscopy and roentgenography (Fig. 6) showed no evidence of herniation into the thorax. The lungs were well aerated and the cardiac silhouette was within normal limits of size and outline.*

Electrocardiographic examination (R. T. Jellison, M.D.) showed sinus tachycardia, P-R interval 0.12, deep S_1, "W" shaped QRS_2, inverted TF_4. The impression was: "The bizarre relation between P and the "W" shaped QRS_2 is possibly based on distortion (rotation and change of axis), but the S_1, Q_3, T_3 is the pattern seen in acute cor pulmonale as in sudden elevation of pulmonary circuit pressure (as in pulmonary embolism)."

At the time of discharge from the hospital February 1, 1946, the incision was healed and the infant weighed 7 lb. 13 oz. On submission of this report for publication, seven months after operation, he was entirely without symptoms referable to the digestive or cardiorespiratory systems and appeared to be in excellent health, with normal weight increase.

The literature affords few references to this condition and careful study of available publications in the Library of the American Medical Association and that of the Surgeon General revealed only 8 previously reported cases. In 2 of these[5] there was a possibility of traumatic etiology. Only 2 were found in infants, and in 1 of these as well as in 2 adults, the abnormality was the proximate cause of death. The diagnosis of diaphragmatic hernia was made during life in only 2 cases.[2,8] In only 1 case[8] was a roentgenographic examination made. In 6 cases[1,2,5,6,7] the hernia was of the false type, i.e. without a sac; in 1 case[5] there was a sac and in the remaining case[8] the type of hernia was not stated. In our case the herniation was of the false type.

DeCardenal and Bourderon[2] described the earliest case in 1903. Their patient was a fifty-eight year old male who was taken ill suddenly with vomiting and colicky abdominal pain. At exploratory operation a peritoneopericardial hernia of the false type, containing the great omentum and a loop of transverse colon was found. The patient died the day following operation.

Martland[6] in 1909, reported the case of a seventy year old male who presented a clinical picture of acute right heart dilatation with cyanosis, dyspnea, rapid irregular feeble pulse, but with no murmurs. At autopsy an oval opening 5 by 6 cm. was found above the liver making a communication between the peritoneum and the pericardium with herniation of the transverse colon and the great omentum. The peritoneum of the under surface of the diaphragm was continuous with the parietal pericardium. There was no hernial sac and no evidence of pathological abnormality beneath the diaphragm.

Keith[5] in 1910 reported 3 cases found at autopsy. The first was in a fifty-eight year old male who had a peritoneopericardial hernia of the false type with contents consisting of a process of the great omentum. The second case also of the false type was in a forty-seven year old male; the hernia contained a process of liver. Congenital nature of the defect was somewhat doubtful in both of these cases since there was a pos-

* On October 17, 1946, approximately nine and one-half months after operation and shortly following submission of this report for publication, a check-up roentgenographic examination was made. At that time the patient was in excellent health, had no symptoms referable to the cardiorespiratory-digestive systems and was gaining weight in normal fashion. This examination disclosed a recurrence of diaphragmatic herniation with a considerable part of the small intestine above the diaphragm. The point of entry was anterior and slightly to the left—approximately the same location as originally. There was no limiting pericardial membrane about the gas and barium-filled bowel and it was concluded that the hernia was of the peritoneopleural type, probably breaking through the incision made in the pericardium at the time of operation. It is proposed to make a further attempt to repair the hernia and this will be reported subsequently.

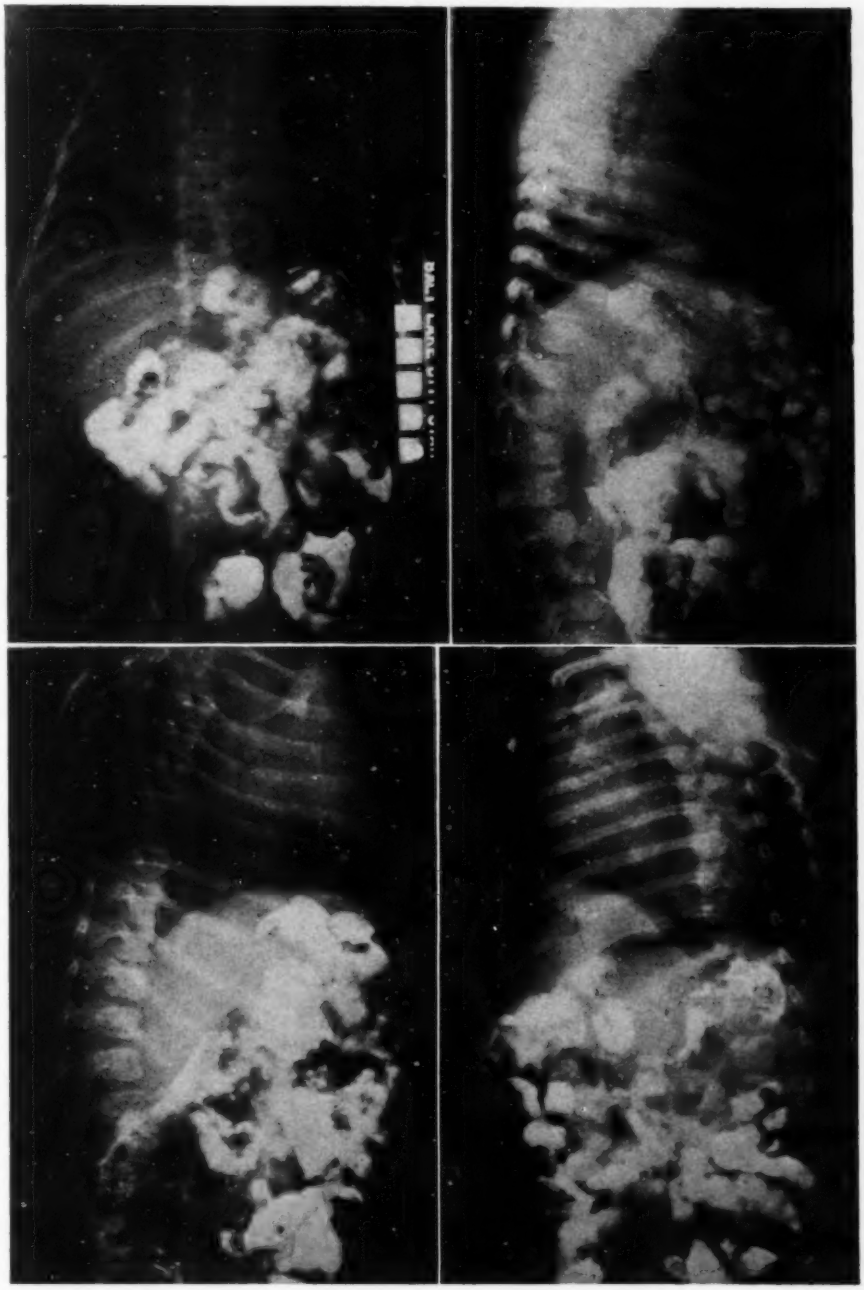


FIG. 6. January 26, 1946, three weeks after operation. Anteroposterior, lateral and oblique studies showing barium-filled intestine beneath the diaphragm and no evidence of herniation into the thorax.

sibility of traumatic etiology. The hernia in the third case, a newborn infant, was of the true type and contained a process of liver.

Taillens,[8] in 1934, described the case of an infant, aged two months, who had shown attacks of cyanosis following meals practically from birth. This was the only case, other than our own, which had a roentgenographic examination, and by roentgenoscopy and film study a diagnosis of diaphragmatic hernia was made, with con-

FIG. 7. Postoperative appearance of chest January 21, 1946, showing cardiac outline and normally aerated pulmonary structures.

tents of hernia consisting of part of the stomach and a portion of small intestine. Because of a rapidly deteriorating condition the patient was not operated upon and died thirteen days after admission to the hospital. At autopsy there was found a circular opening about 3 cm. in diameter between the peritoneal and pericardial cavities. The hernia contained the pyloric half of the stomach, the duodenum, part of the left lobe of the liver and almost all of the large intestine. The heart was displaced to the right and posteriorly. The type of hernia was not given.

O'Brien[7] in 1939, reported the case of a sixty-four year old male autopsy specimen having an aperture 2 by 2.3 cm. in the anterior leaf of the centrum tendineum where it is fused to the floor of the pericardium. It was well to the left of the falciform ligament of the liver and anterior to the left part of the coronary ligament. The pericardium was continuous with the peritoneum all around the margins of the aperture, and a small process of liver protruded about 4 mm. into it. He also found 4 cases in the dog and 1 in the ass.

Casey and Hidden[1] in 1944 reported the case of an infant who died four hours after birth. At autopsy it was found that, among other anomalies, there was absence of the central tendon and anterior portion of the diaphragm with the presence of a pericardioperitoneal foramen, but abdominal contents had not herniated through it.

Giffen,[3] 1912, in a review of cases of diaphragmatic hernia at the Mayo Clinic, stated that herniation of abdominal contents into the pericardial sac had been reported but gave no reference to it. Hedblom[4] listed 1 case but did not describe it or give references to the literature.

Inquiry of the Library of the American Medical Association revealed no additional cases since the beginning of 1946.

According to Martland[6] the defect is determined by the fifth week of embryonal development. In the two weeks embryo the primitive heart is formed by coalescence of two tubes produced in the splanchnic mesoderm, and it subsequently becomes attached to the anterior and posterior body walls by ventral and dorsal processes known respectively as the mesoderm anterius and mesoderm posterius. Later the vitelline and umbilical veins in the ventral and lateral aspects of the body diverge to reach the heart, and in so doing, carry with them a layer of the mesoderm in which they are embedded. These folds approach and finally meet the mesoderm anterius to form the septum transversum, which is the anlage of the future diaphragm. In the next phase the pericardial and pleural cavities are closed off by development of the pleuropericardial fold which is formed by an inwardly projecting membrane containing the ducts of Cuvier. It would appear, therefore, that a peritoneopericardial hiatus depends upon failure of fusion of the septum transversum and also of the pleuropericardial fold, leaving a communication between the peritoneal and pericardial cavities. And furthermore, that the condition is determined early in embryonal life since both of the phases in which development is defective should be complete by the fifth week of gestation.

SUMMARY

A rare case of congenital peritoneopericardial defect with herniation of abdominal contents into the pericardium is reported. The patient survived operation for repair and at the time of this report was in excellent health. Only eight other cases have been found in a careful search of the literature. Two of these eight cases were operated upon but failed to survive. Five cases, discovered at autopsy, had no symptoms referable to the condition during life, hence it would appear that the defect is not inimical to life. As far as we have been able to discover, an accurate diagnosis has not been made prior to operation or autopsy examination.

Clinically the condition cannot be distinguished from the more common peritoneopleural diaphragmatic herniae since both types have predominating symptoms of varying degrees of dyspnea and cyanosis following feedings, with reduction of intensity of these symptoms after vomiting. However, we feel that the possibility of peritoneopericardial herniation should be thought of and investigated in all cases showing cardiorespiratory embarrassment, but especially those having post-prandial cyanosis relieved by vomiting.

Only one other case was investigated roentgenographically.[8] While in it and in ours a diagnosis of diaphragmatic hernia was made during life, the exact type was not identified chiefly because of lack of knowledge of possible existence of such abnormality. The diagnostic roentgen features of the condition appear to be:

(a) The presence of opacity in the thorax, not accounted for by normal structures, which may give the appearance of an enlarged heart or of congenital pulmonary atelectasis.

(b) The presence of sharply defined convex borders, representing the distended pericardium, about the opaque area should arouse suspicion and stimulate further investigation.

(c) The diagnosis may be made with practical certainity if coils of intestine can be identified by gas or opaque medium content to obscure the cardiac shadow and to lie within the confines of the distended pericardium.

Angus K. Wilson, M.D.
343 South Main St.
Salt Lake City, Utah

REFERENCES

1. Casey, A. E., and Hidden, Eleanor H. Nondevelopment of septum transversum, with congenital absence of anterocentral portion of the diaphragm and of the suspensory ligament of the liver and presence of an elongated ductus venosus and a pericardioperitoneal foramen. *Arch. Path.*, 1944, *38*, 370–374.
2. DeCardenal, Grenier and Bourderon. Hernie diaphragmatique du grand epiploon et d'une anse du colon transverse dans le pericarde chez un adulte. *J. méd.*, Bourdeaux, 1903, *23*, 222–224.
3. Giffen, H. Z. Diagnosis of diaphragmatic hernia. *Ann. Surg.*, 1912, *55*, 388–397.
4. Hedblom, C. A. Diaphragmatic hernia; study of 378 cases in which operation was performed. *J.A.M.A.*, 1925, *85*, 947–953.
5. Keith, A. Three cases of peritoneo-pericardial diaphragmatic hernia. *Brit. M. J.*, 1910, *2*, 1297–1298.
6. Martland, H. S. A case of congenital false diaphragmatic hernia. *J.A.M.A.*, 1909, *20*, 1574–1576.
7. O'Brien, H. D. Pericardio-peritoneal communication. Description of a rare type of diaphragmatic hernia. *J. Anat.*, 1939–1940, *74*, 131–134.
8. Taillens, J. Un cas rare de hernie diaphragmatique congénitale. *Bull. Soc. de pédiat. de Paris*, 1934, *32*, 558–560.

UNUSUAL VENOUS RETURNS OF THE LOWER EXTREMITIES

By E. C. BAKER, M.D.
Department of Radiology, Youngstown Hospital Association
YOUNGSTOWN, OHIO

THE venous system of the lower extremity is essentially a large plexus with two main divisions, one superficial and one deep. These two divisions have free intercommunication in the leg and thigh. The blood from these plexuses is gradually collected into main physiological channels. For the superficial portion of the system the internal saphenous vein is the normal main channel. In the leg the tibial veins are the chief collecting channels for the deep plexus. These two veins and the peroneal vein, when seen, unite below, in the region of, or above the knee to form the popliteal vein. The popliteal vein at the upper portion of the popliteal space beomes the femoral vein. This is the main collecting channel for the anterior and inner portion of the thigh. In the fossa ovalis the internal saphenous vein from the superficial system and the femoral vein from the deep system unite to form the external iliac vein. This briefly outlines the venous channels which are visualized in the normal venogram of the leg and thigh.

The venous blood from the posterior portion of the thigh and some of the outer portion of the thigh returns through a vein accompanying the inferior gluteal artery. This vein finally empties into the internal iliac vein within the pelvis. This particular portion of the venous system of the lower extremity is not ordinarily visualized by the usual venogram.

In the normal case dye injected in the region of the foot or below the ankle normally progresses upward and gradually inward to the main physiological channels unless a block occurs. Occasional cases have been seen where retrograde flow of the dye does occur through normal valves and without block proximal to the area of the retrograde flow. Where block occurs in pathological material retrograde extension of the dye through valves is commonly seen. Where block of venous return is demonstrated the dye will go through any part of the venous plexus which is patent and which allows the dye to proceed around the block. Usually when the dye reaches an area above the block it tends to return to the normal physiological main channels.

The unusual returns which will be described have been seen in pathological material. Acute block of the main deep physiological channel of the thigh has been well described by Bauer[2] in his original monograph and has since been described by Lesser and Danelius[3] and briefly by the author with Sedwitz.[1] In the acute block the dye seeks numerous small veins below the site of the block. These veins form a lacy network in the superficial tissues. In these cases the dye is usually lost before main physiological channels are reached above the block. The roentgen picture is that of a lacy network which extends gradually upward in the superficial tissues and fades out as the dye spreads into smaller and smaller veins of the superficial plexus. This lacy network may be seen in the mid-thigh or in the upper thigh or extending around toward the buttock depending on the area of the block of the main physiological channel. This particular pattern once seen is easily recognized.

In Figure 1 the femoral vein in the upper thigh is not visualized. The internal saphenous vein is visualized upward to the region of the fossa ovalis. Numerous small veins in the superficial tissues of the upper thigh and perineum can be seen. These veins are gradually lost as the dye progresses away from the internal saphenous. In this case none of the dye is seen entering the external iliac. The block involves the

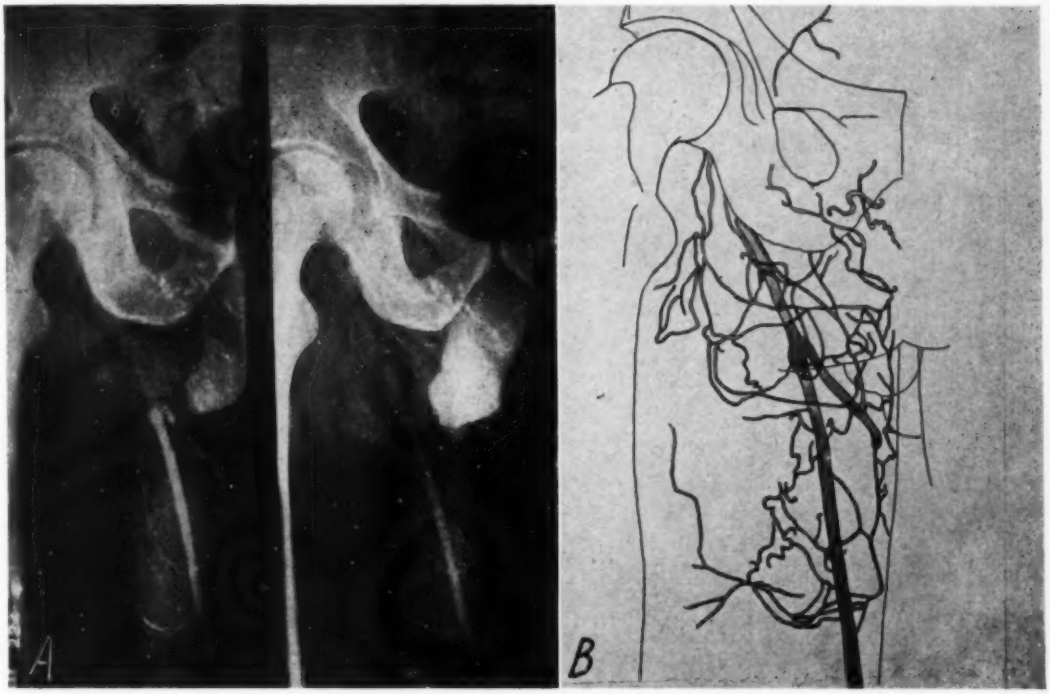


Fig. 1. *A*, acute block of deep circulation of the thigh.
B, drawing from original roentgenogram.

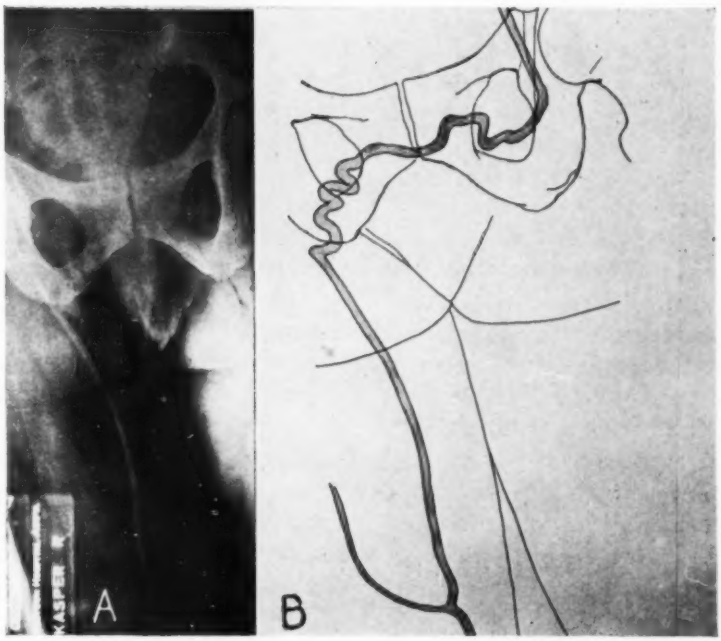


Fig. 2. *A*, chronic block of deep circulation of the thigh with return through the pudendal circulation. *B*, drawing from original roentgenogram.

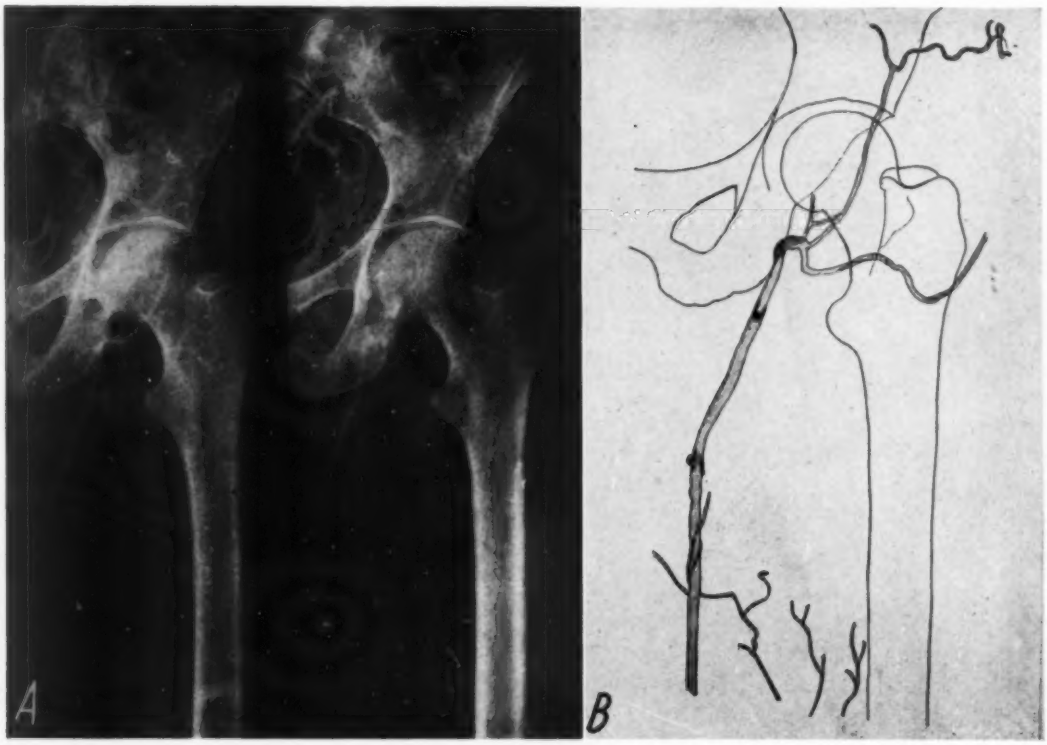


FIG. 3. *A*, chronic deep block with return through circumflex veins. *B*, drawing from original roentgenogram.

deep circulation of the thigh and the external iliac vein beyond the fossa ovalis. This particular case had an embolic process in the lung fields.

Following an acute block such as illustrated by Figure 1, nature gradually finds larger physiological channels in the area of the block so that in the matter of a few weeks to several months the small venous plexus carrying the load in the acute block is no longer visualized. Certain main physiological channels are formed out of already existing veins and these become the main channels for the blood stream in these blocks of long duration. These main physiological channels seem to be formed from the previous larger veins in the plexus. In the region of the fossa ovalis and from there upward these veins go into the body by means of several routes. It is usually possible to identify these veins by their course and location. Usually, however, the blood flow through these veins is now reversed and the dye extends outward from the region of the fossa ovalis through these veins to collateral circulation.

In Figure 2 the result of a chronic block is demonstrated. No deep circulation is demonstrated in the upper thigh. The dye is returning through the internal saphenous vein to the region of the fossa ovalis. In this region the dye comes out of the internal saphenous into a fairly large and tortuous vein in the pudendal area. This large and tortuous vein crosses through the pudendal area from the affected side to the opposite side. The dye then comes in through the pudendal circulation of the opposite side to the region of the fossa ovalis, enters the circulation of the opposite leg at the fossa ovalis and can be seen going upward through the external iliac of the opposite side. In this case the dye has gone retrograde through the pudendal circulation of the affected side and then gone normally through the pudendal circulation of the

opposite side into the external iliac vein.

In Figure 3 again the deep circulation of the thigh is missing. The dye is shown in the internal saphenous vein to the region of the fossa ovalis. In this area the dye comes outward into circumflex veins which circle anteriorly in the superficial tissues of the thigh and laterally toward the buttock. The dye can be traced around into the area of the buttock. Here again the collateral circulation which is taking up the load following the deep block is a vein which normally carries the blood stream in the opposite direction. Normally these veins collect the blood from the lateral portion of the thigh and buttock and bring it around to the internal saphenous vein close to the fossa ovalis. In this case, however, the dye is spreading retrograde through these veins into the veins of the buttock.

In Figure 4 most of the dye is returning from the lower leg through the internal saphenous. A small vein can be seen coming up in the region of the posterior thigh. At the fossa ovalis the internal saphenous is blocked. The femoral vein is not seen. The dye comes out of the internal saphenous into pudendal veins which turn inward and anastomose with veins of the perineum. In the region of the sciatic notch, this vein enters the body and can be seen going upward irregularly in the posterior portion of the pelvis. On the original roentgenogram the dye can be traced into a mid-sacral vein immediately anterior to the sacrum and in this crosses toward the opposite side of the body. Here another area of collateral circulation is used to return the dye and blood to the body.

In Figure 5 another type of return is demonstrated. A portion of the dye from the lower extremity returns in the internal saphenous vein to the area of the fossa ovalis. In this area a small amount of dye enters the femoral vein and extends retrograde in the femoral vein for a distance of 6 to 8 inches. Below this point the femoral vein was not visualized. In the leg no evidence of deep circulation was demonstrated. Starting in the leg and coming up to the lower thigh there is an irregular vein seen in the posterior portion of the leg, knee and thigh. This appears to be carrying the greater part of the dye. It can be seen as a very tortuous vein immediately below the head of the femur and extending upward through the region of the head of the femur. Stereoscopically this vein can be demonstrated to run posterior to the head of the femur and through the sciatic notch

Fig. 4. Chronic deep block with return through perineal veins and mid-sacral veins.

into the posterior portion of the pelvis. Here again, the veins which are already in existence appear to be utilized. The dye comes upward through superficial veins to the upper thigh. At this point it comes in more deeply. It reaches the vein that follows the inferior gluteal artery and goes upward through this into the posterior pelvis toward the internal iliac. This particular portion of the circulation is seen only in cases of block and is not normally visualized in the venogram of the leg and thigh.

In Figure 6 the deep circulation of the thigh is not visualized. The internal saphenous vein is not demonstrated. The

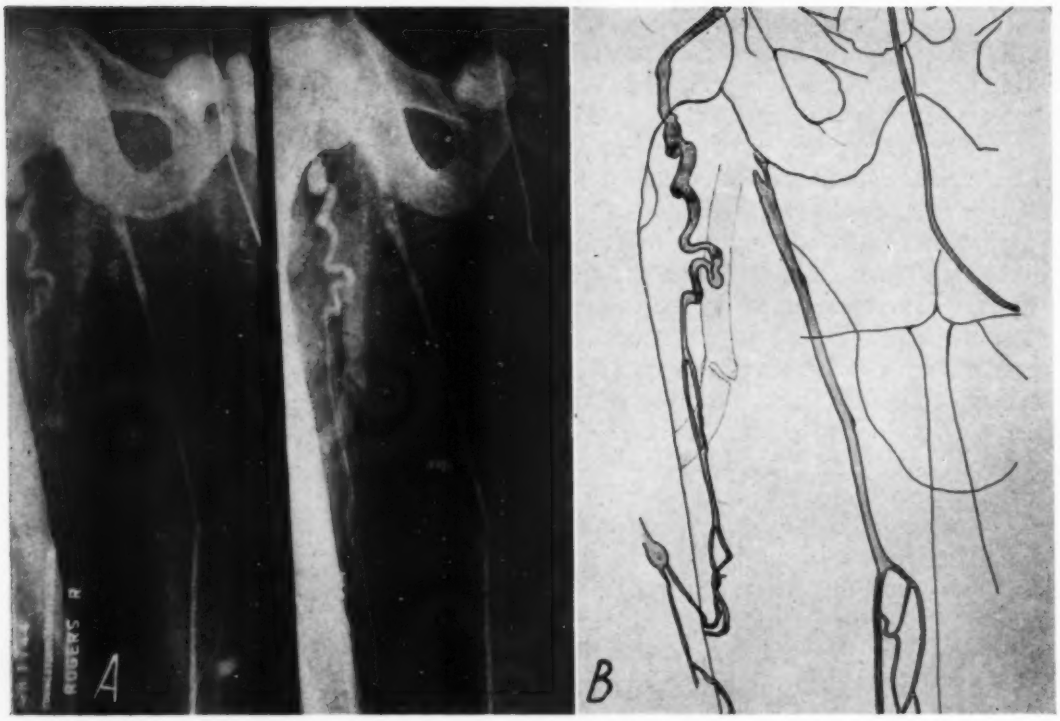


FIG. 5. *A*, block of the circulation in the region of the fossa ovalis, partial return through the inferior gluteal vein. *B*, drawing from original roentgenogram.

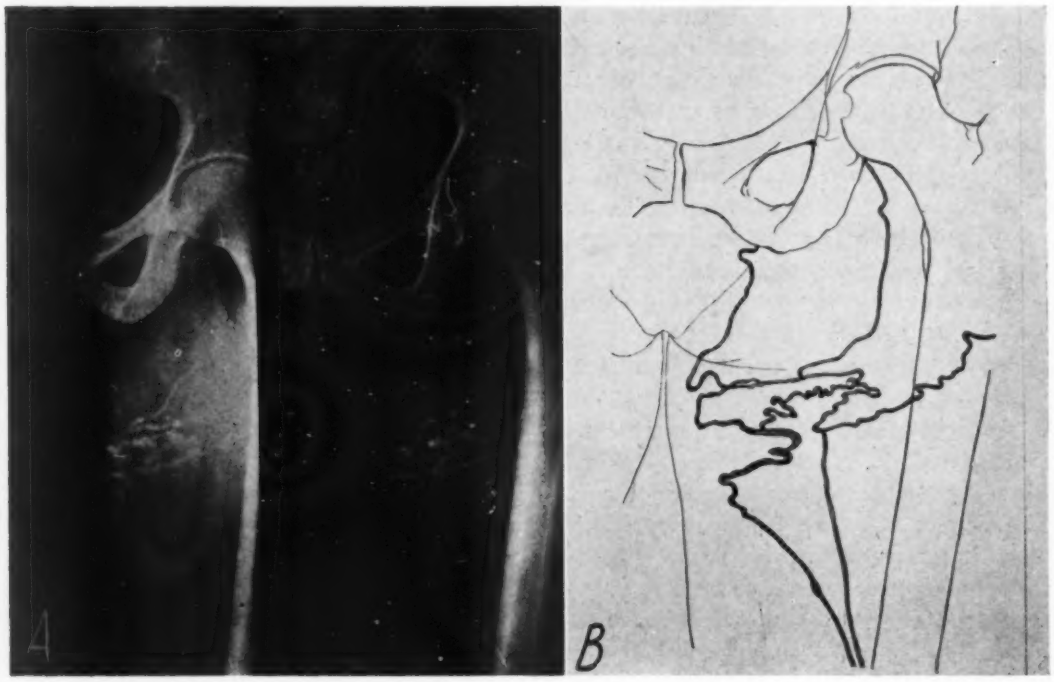


FIG. 6. *A*, block of deep and superficial circulation in the thigh, return through pudendal vein and inferior gluteal vein. *B*, drawing from original roentgenogram.

dye is returning upward through a tortuous and very irregular circulation that lies first in the posterior portion of the superficial tissues of the thigh and then spreads through the inner aspect of the thigh in the superficial tissues. Close to the pelvis one small branch can be seen coming upward to the region of the pudendal circulation and another branch is demonstrated running upward toward the head of the femur. This, on stereoscopic localization, extended backward into the region of the vein accompanying the inferior gluteal artery and so into the pelvis.

These cases are presented as types of unusual venous return which have been seen on a number of occasions. Two observations should be made. When unusual types of return are visualized in the region of the fossa ovalis or upper thigh such returns are, I believe, conclusive proof of deep block. Second, these returns tend to follow already existing main channels in a retrograde fashion until normal upward channels are reached.

Youngstown Hospital Association
Youngstown 1, Ohio

REFERENCES

1. Baker, E. C., and Sedwitz, S. H. Observations on venography of lower extremities. *Radiology*, 1943, *41*, 451–458.
2. Bauer, G. Venographic study of thrombo-embolic problems. *Acta chir. Scandinav.* (suppl. 61), 1940, *84*, 1–75.
3. Lesser, A., and Danelius, G. Venography; its value in diagnosis and management of venous disturbances of lower extremities. *Ann. Surg.*, 1944, *119*, 903–921.

OSTEOCHONDRITIS DISSECANS*

AN ANALYSIS OF FORTY-TWO CASES AND A REVIEW OF THE LITERATURE

By MAJOR GERALD LAVNER
MEDICAL CORPS, ARMY OF THE UNITED STATES

OSTEOCHONDRITIS dissecans has been defined as a non-infectious aseptic necrosis of a segment of subchondral bone of the long bones of the extremities, resulting ultimately, although not invariably, in an osseocartilaginous sequestrum into the affected joint.[14]

Although mention is made by some of the occurrence of this lesion in the head of the humerus, the acetabulum, the calcaneus and carpal navicular and lunate bones, no cases are actually described and no definite references have been made by these authors, nor could such case reports be found in the available literature.

A review of the subject has been undertaken on the basis of 42 cases occurring in various locations, seen at Lovell General Hospital in a two year period. Twenty-three of these cases were proved by operation. The roentgenologic and other data of the remainder were so characteristic that they have been included in this analysis.

The condition was recognized as early as 1736 by Alexander Munro,[20] but the term, osteochondritis dissecans, was first employed by König[15] in 1888.

ETIOLOGY

The pathogenesis of osteochondritis dissecans is not clearly established. Many theories have been proposed and thoroughly discussed.[22,27] Briefly, these theories may be divided into the traumatic and non-traumatic. Undoubtedly direct trauma, with partial or complete separation of a portion of bone and cartilage is the causative factor in some cases. This, however, fails to adequately explain the production of sequestra in cases in which a complete history fails to elicit a story of trauma. The familial tendency and the cases of bilateral involvement, as reported by Bernstein,[4] remain unexplained. Furthermore, since the osteochondritic site in the knee is well protected in all positions of the joint and since the roentgen appearance is not that of a fissure fracture, it is unlikely that direct trauma is the immediate factor in a majority of cases. There was a history in 25 of our 42 cases to warrant consideration of trauma as a significant etiologic factor. König suggested that an inflammatory lesion, possibly produced by trauma, resulted in the occlusion of a terminal artery supplying an area of articular cartilage. The sequestrum thus formed became detached and subsequently was extruded into the joint. With reference to the knee, Freiberg[9] and Fairbank[8] postulated that in many instances primary arteriolar occlusion was precipitated by the impingement of an elongated medial tibial spine on the articulating surface of the medial femoral condyle, the most frequent site of involvement. Only one of our cases (Fig. 1) showed an elongated spine. The importance of an elongated medial tibial spine in the production of this lesion is therefore open to question. Axhausen[2] has stated that non-traumatic occlusion of an epiphyseal artery by an embolus, with resultant aseptic necrosis, might be the causative factor in some cases. The changes which result simulate the process seen in Legg-Perthes' and in Kienböck's disease.

Of these many theories which have been proposed, that of trauma, either direct or indirect, is generally accepted, inasmuch as a definite history of antecedent injury can be obtained in a majority of cases. In the 42 cases which we have observed during the

* From the Roentgenologic Service of Lovell General Hospital, Fort Devens, Mass.

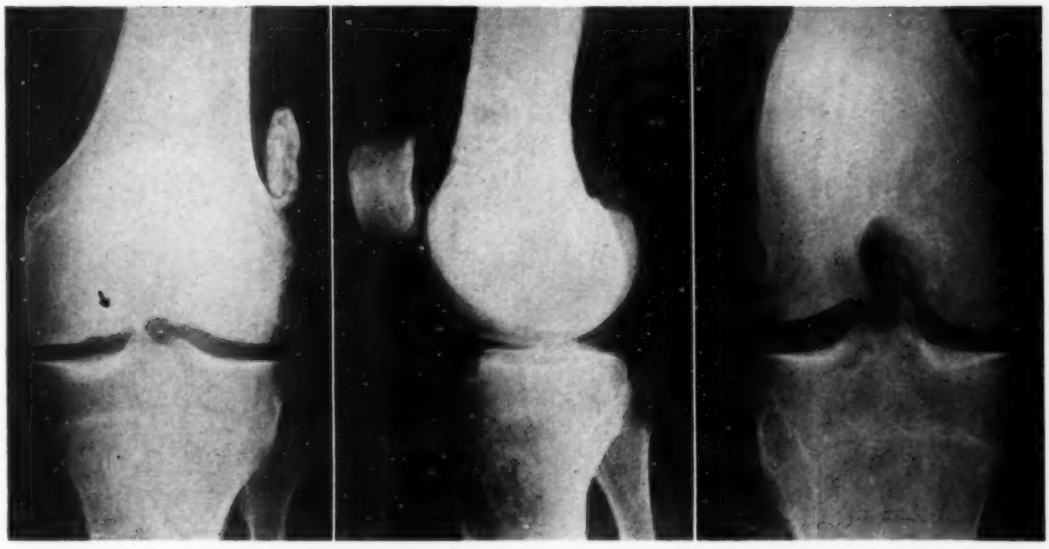


Fig. 1. Osteochondritis dissecans of the medial femoral condyle (arrow). Another large laminated loose body may have arisen from the same site and grown by accretion. Note the elongated medial tibial spine. Believed to play an etiologic rôle, this occurred only once in the present series. The arthritic changes which may result from a lesion of long standing are well shown.

past two years, a history of trauma was elicited in approximately 60 per cent. Osteochondritis dissecans is a disease of youth. The exact length of time required to produce a sequestrum is not known, but it is possible that injuries sustained in childhood and minimized at that time may be responsible, since the lesions seen in our cases with recent histories of trauma are too far developed, it seems, for trauma to have been a causative factor.

PATHOLOGY

Knowledge of the pathology which occurs in osteochondritis dissecans has previously been obtained entirely from observations made at the time of operation, and from the microscopic examination of fragments which were removed. Recently we have had the unusual opportunity to study at postmortem a case of osteochondritis dissecans affecting the medial condyle of the femur. The condition had been diagnosed by roentgen examination approximately five months before the time of death (Fig. 2*a*). At postmortem the knee joint was opened and inspected. The lesion occupied its usual site on the medial femoral condyle and was found to be well advanced, since the characteristic changes of flattening of the affected area and fragmentation of the sequestrum were well demonstrated (Fig. 2*b*). The cartilage around the margin of the defect was incised and the sequestrum separated very easily (Fig. 2*c*). A block of bone which included all of the diseased area was excised for pathologic examination. It can readily be seen that the gross characteristics of the specimen excised conform exactly to those features of the disease which have previously been observed at operation and described in the literature. The sequestrum lies in a bony recess and is held in place by intact overlying articular cartilage and synovial membrane. Exceptionally the recognition of the site of osteochondritis dissecans may be difficult as the changes in the articular cartilage may not be advanced enough to permit easy recognition. At times the only evidence is the presence of a slight groove in the overlying cartilage. Later the sequestrum is demarcated from its bed by a layer of thin fibrous tissue which facilitates recognition of the condition. When freely movable in the articular space, the

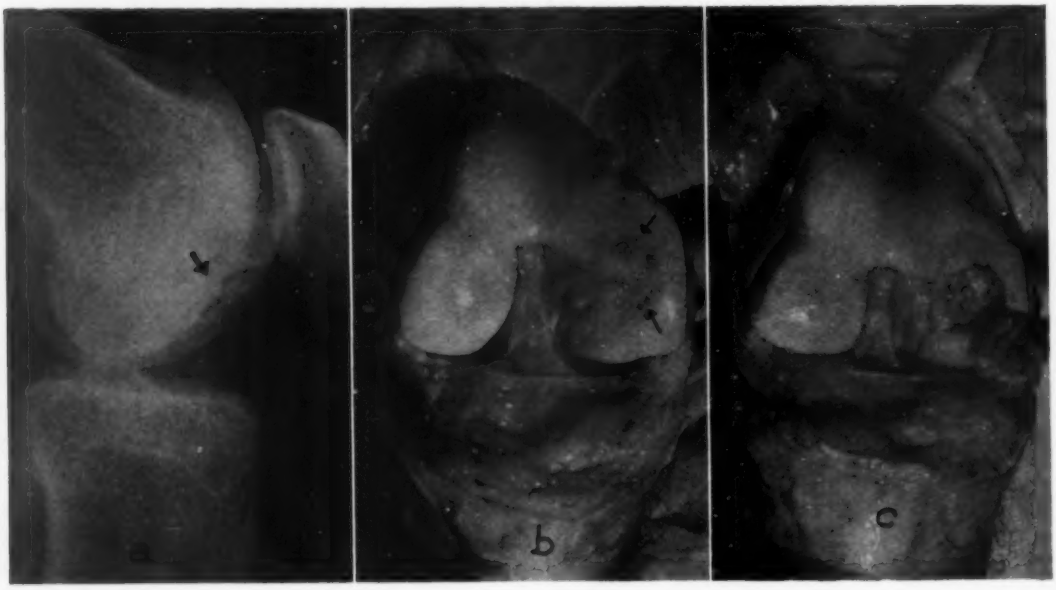


FIG. 2. (*a*) Lateral roentgenogram of the knee made five months before death shows a fragmented sequestrum in a defect in the medial femoral condyle (arrow). (*b*) Knee joint, opened at postmortem, shows the slight groove in the articular cartilage of the medial femoral condyle (arrows). (*c*) The grooved cartilage was incised and the button of bone separated.

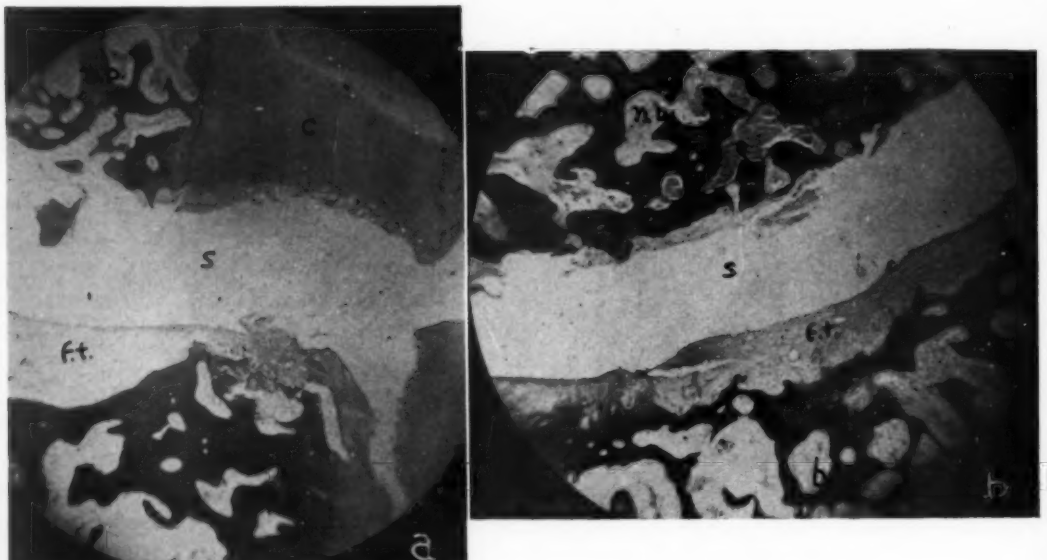


FIG. 3. (*a*) Low power projection shows necrotic bone (*n.b.*) covered by cartilage (*c*). The femoral condyle is covered by fibrous tissue (*f.t.*). The space (*s*) is an artefact. (*b*) High power: Sequestrum (*n.b.*) is separated from the femoral condyle which shows normal bone (*b*) covered by fibrous tissue (*f.t.*) at the site of the lesion. The space (*s*) is an artefact.

fragment tends to become rounded and to increase in size by the accretion of cartilage and new bone. In some instances it may break up into several fragments. Many authors attribute to Raney the statement that the fragments which become adherent to the synovial lining of the joint continue to grow.

Fig. 4. Bilateral medial femoral condyle lesions. The defect in each medial femoral condyle is almost invisible on the anteroposterior views, and barely visible on the lateral views (arrows). The "tunnel" views are surprisingly informative. Note the slight intercondyloid displacement of the sequestrum on the right. The patient complained of symptoms on this side only.

Microscopic examination of the osteochondritic focus reveals that the center is formed by necrotic bone with loss of lamellar substance, or by an irregular mass of ossifying cartilage with foci of necrosis at its periphery (Fig. 3). The picture is one of necrosis with low grade, aseptic, productive inflammatory change. A thick layer of hyaline cartilage is present on the articular surface, and a thin layer of fibrocartilage on the other.

Incidence and Sites of Occurrence

Osteochondritis dissecans primarily affects young adult males. With reference to its occurrence in the hip, King and Richards[14] found that of 28 cases, 26 occurred in males. In the series reported by Phemister,[22] males predominated in the proportion of 13 to 1. In an analysis of 153 patients with knee injuries, exclusive of fractures, admitted to a Hospital Ship in fourteen months, Allan and Nicholson[1] found 5 cases of osteochondritis dissecans. This condition has been more frequently recognized in the military service then heretofore. This may be due to an aggravation of symptoms by physical activity incident to military service and to the increased availability of roentgenographic examination to a large number of youths.

Of the various bones which may be in-

FIG. 5. Osteochondritis dissecans of the glenoid of the scapula. The site of origin is evident in the lower portion of the glenoid (black arrow). The sequestrum is displaced into the shoulder joint (white arrow).

volved, the femur, and particularly its medial femoral condyle, is affected in about 85 per cent of the cases.[7] Cases with involvement of the lateral femoral condyle have been reported.[3,11] The other bones which are less frequently affected, as shown by occasional case reports, include the femoral head,[23] the capitellum,[21] and the supratrochlear septum[19] of the humerus, the radius,[12] the talus,[6] the metatarsal bones,[5] the phalanges,[11] the patella,[24] the metacarpal bones,[11] and in one of the tarsal cuneiforms.[10]

In a two year period at this Army General Hospital 891 knees were examined roentgenographically for conditions other than fracture or gunshot wounds. A total of 33 cases of osteochondritis dissecans was found among these. In 27 cases the medial femoral condyle, in 2 the lateral femoral condyle, and in 4 the patella was involved. In 1 case there was bilateral involvement of the medial femoral condyles (Fig. 4). In his series, King[13] reported bilateral involvement in 20 per cent.

We have also seen 1 case involving the glenoid of the scapula, 1 case in the proximal phalanx of the great toe, 1 case in the talus, 3 cases involving the supratrochlear septum of the humerus, and 2 patients with bilateral lesions of the head of the radius. With the exception of the talus, none of these cases has been proved by operation but the roentgenologic appearance is so characteristic that the diagnosis seems incontrovertible. A review of the available literature failed to disclose a case similar to that seen in the scapular glenoid (Fig. 5) or involvement of the other bones of the shoulder girdle by osteochondritis dissecans. Hutchison[11] reported a case of osteochondritis dissecans occurring in the base of the proximal phalanx of the great toe, also unproved by operation. He failed to find any similar case recorded in the literature before 1943. We have seen 1 case involving the proximal phalanx of the great toe (Fig. 6). This patient was not operated upon.

Briefly, the typical locations of this disease are: (1) in the knee—most often the mesial femoral condyle, rarely the lateral; (2) in the hip—on the superolateral aspect of the femoral head; (3) in the elbow—the capitellum and supratrochlear septum of the humerus, and the head of the radius; (4) in the ankle—the superomedial aspect of the talus, rarely the superolateral aspect; (5) in the patella—the posterior articulating surface, usually of the lower pole; (6) in the first metatarsal bone—the head; (7) in the phalanges—at the proximal end.

SYMPTOMATOLOGY

The symptoms of osteochondritis dissecans are dependent upon the site of involvement and the stage of sequestration. It is not unusual to find a well demarcated, undetached sequestrum as an incidental finding in the roentgen examination of extremities or joints where recent fracture is suspected, or where a "normal" extremity is taken for comparison. King[13] has chosen to call these "slumbering" cases. In the knee, before the sequestrum has become detached, the usual complaints are pain or

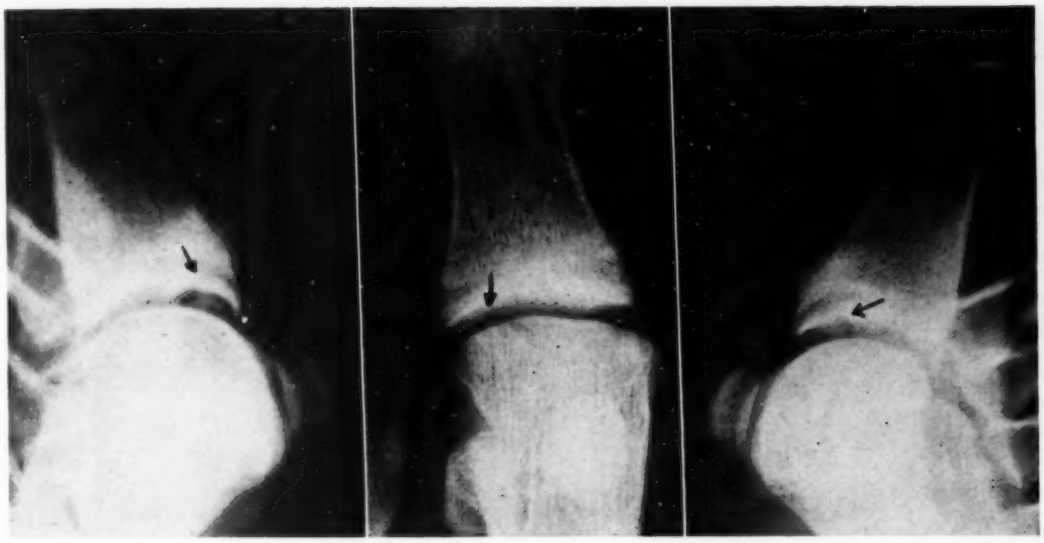


Fig. 6. Osteochondritis dissecans involving base of proximal phalanx of great toe (no operation). The characteristic defect and the included sequestrum (arrow) are well shown. This is a rare site for this lesion.

stiffness occurring after marching, prolonged standing or unusual physical exercise. In some cases this may be accompanied by swelling of the joint. After the sequestrum has become detached, the usual story elicited is that of repeated sudden "locking" of the joint, similar to that occurring with loose joint bodies from other causes. In some instances the patient has learned to disengage the fragment and is therefore often able to carry on his normal duties. If the sequestrum becomes adherent to the joint capsule, the locking may disappear.

In the sites less frequently affected, the symptoms of slight pain or lameness after trauma are noted. A history of antecedent sprain may be elicited in cases involving the ankle. Frequently there is limitation of motion in the elbow, and locking may occur.

Objectively, the signs are usually few in number. The affected joint may reveal either nothing abnormal, or may show only slight swelling and limited motion. Later, wasting of muscles may occur, and rarely, the loose bodies may be palpable.

To summarize, chronic pain and discomfort after trauma, accompanied by instability of the joint, or "locking," should suggest the possibility of osteochondritis dissecans.

ROENTGENOLOGY

The roentgenographic appearance of osteochondritis dissecans is characteristic and should be unmistakable. Characteristically, the articular surface of the involved bone shows a concave defect one or more centimeters in diameter. The margins are usually smooth but may be slightly wavy. Rarely the margins are undercut irregularly. Within the concavity, but distinctly separated from its margins, is an ovoid, dense, sharply marginated, usually homogeneous, bony fragment. In some instances the sequestrum is fragmented. Occasionally the defect appears disproportionately large for the sequestrum within it due probably to separation and displacement of one of the fragments. In some cases, the concavity is shallow with long, sloping margins, suggesting an attempt at healing during a long period of time. Infrequently the loose detached fragment is much larger than its site of origin, suggesting that in its separated state it has become larger by accretion of bone. Sometimes, in long-standing cases, marked osteophyte formation is

present to obscure the bony defect and a careful search is necessary to locate the site of origin of the loose body.

In the knee, Liebman and Iseman[16] believe that the lesion of osteochondritis dissecans is best seen in the anteroposterior roentgenogram, and is likely to be overlooked in the lateral view. In our experience, the lesion is much more easily demonstrable in the lateral projection. This is of no great importance, since routinely our examination of the knee consists of three views: the anteroposterior, the lateral, and the posteroanterior "tunnel" view. Frequently, this last projection is of the greatest aid in demonstrating the sequestrum and the bony defect in the intercondyloid region of the femur. To obtain the tunnel view, the patient lies in the prone position with the knee flexed at 45 degrees. The central ray is projected through the knee joint, tangential to the superior surface of the tibial condyles. By this procedure, loose bodies which might ordinarily be obscured are projected into the intercondylar space and the bony defect in the condyle may be better visualized.

In 27 of the cases in the knee, the intercondyloid aspect of the medial femoral condyle was the most frequent site of the osteochondritic focus. It is interesting to note that 50 per cent arose from the central portion of the articulating surface of the condyle. The remainder were equally divided between the anterior and posterior aspects.

In only 2 cases was the lateral femoral condyle the site of the disease. The defect was much smaller and shallower than was ordinarily observed in the lesions occurring in the medial condyle. The condyle was irregularly flattened, the flattening extending to the lateral margin of the bone in a step-like fashion (Fig. 7).

In the patella, the site of the disease may be recognized as a defect in the otherwise smooth, slightly concave, posterior surface, usually at the lower pole. The defect is small, measuring 3 to 7 mm. in diameter. The sequestrum is somewhat smaller, and,

FIG. 7. Osteochondritis dissecans in the lateral femoral condyle. This case was proved by operation. Note the small steplike defect (single arrow). The other case of lateral femoral condyle lesion in our series presented a similar appearance. An area of ossification due to Pellegrini-Stieda's disease is present adjacent to the internal femoral epicondyle (double arrow).

Fig. 8. (*a*) Case III. Osteochondritis dissecans of the patella. A long shallow depression is noted in the articulating surface of the lower pole of the patella (upper arrow). In the upper part of this depression is seen a small separated fragment of bone. Another loose body is seen in the intercondyloid portion of the knee joint (lower arrow). This also arose from the patellar defect. This case was proved by operation.

(*b*) Case IV. Osteochondritis dissecans of the patella. There is a small defect with osteoporosis about it at the inferior pole of the patella (long arrow). A small fragment of bone is loose in the knee joint close to the femur (retouched). The small bone fragment separated from the lower pole of the patella but close to it (two short arrows) is due to a congenital anomaly; at operation it was found imbedded in the patellar tendon.

because of its size, may cast only a faint shadow (Fig. 8). The tangential view, either inferosuperior or superoinferior, may be of great aid in discovering a lesion at this site. To obtain this, the patient lies prone with the knee fully flexed and the central ray is directed perpendicularly between the patella and the femur, or with the patient supine and with the knee slightly flexed, the ray is directed towards the feet to obtain the same projection.

In the elbow, there have been cases reported with involvement of the capitellum,[9,21] the supratrochlear septum of the humerus,[19] and the head of the radius.[12] The capitellum has been considered to be the most frequent site of involvement in this region. The osteochondritic focus, which usually appears on its posterior aspect, does not differ in appearance from the characteristic lesion described in the knee. Three cases of osteochondritis dissecans of the supratrochlear septum of the humerus have been encountered in our series. The supratrochlear septum is the name applied by Morton and Crysler[19] to that portion of the distal end of the humerus which separates the coronoid anteriorly from the olecranon fossa posteriorly. It usually varies from 0.5 millimeter to 1 centimeter in thickness. In some cases the bony plate becomes absorbed and a supratrochlear foramen results. When the elbow is fully flexed, the coronoid process of the ulna fits into this portion of the humerus anteriorly, while on complete extension the olecranon process impinges posteriorly. Morton and Crysler reported 6 cases, 5 of which were confirmed by operation. They state that these are the first published cases of osteochondritis dissecans occurring in the supratrochlear septum. Two of our cases showed small bony fragments lying within the supratrochlear foramen. There was no demonstrable cup-shaped defect to denote the site of origin. In the third case (Fig. 9) characteristic, well circumscribed sequestra were present both anteriorly and posteriorly, along with typical defects in the supratrochlear septum.

The characteristic lesion of osteochondritis dissecans is occasionally observed in the head of the radius. We had one patient, a colored male, aged nineteen, with lesions in the head of each radius. This patient shows signs of some endocrine dysfunction, as evidenced by delayed epiphyseal closure. In this patient (Fig. 10), the sequestra are slightly displaced. The deformity of the involved portion of the radial head on each side is marked. There is a flattened, somewhat concave defect in the radial head from which arises an ovoid dense sequestrum, similar to those seen in the characteristic lesions in other bones. This patient is awaiting operation because of considerable disability and limitation of motion of the affected joint.

FIG. 9. Case v. Osteochondritis dissecans of the supratrochlear septum of the humerus. The large sharply defined sequestrum lies within a sharply marginated cavity posteriorly (two short arrows). A smaller lesion of similar nature lies anteriorly (double arrow).

Although it has been stated[26] that osteochondritis dissecans occurs in the shoulder, especially in the head of the humerus, we have not seen a case with involvement of this site. A patient who showed a characteristic translucent defect in the articular surface of the glenoid of the scapula and a loose body in the shoulder joint (Fig. 5) was observed. Although unproved, this case fulfills precisely the necessary criteria for a diagnosis of osteochondritis dissecans.

In the ankle, osteochondritis dissecans usually involves the anteromesial aspect of the superior articulating surface of the talus. Occasionally, it may occur on the anterolateral aspect. Except for being smaller, the appearance of the lesion does not differ from that seen in the medial femoral condyle.

Oblique views are very useful in demonstrating lesions of both the ankle and elbow. Over-penetrated films or those made with the grid diaphragm are also often extremely helpful. Mensor and Melody[17] used tomography to clarify the diagnosis in cases involving the ankle joint.

In summary, the characteristic roentgenographic appearance of osteochondritis dissecans is an articular defect with a more or less matching sequestrum, depending on whether the separated fragment is in situ or detached. The roentgenographic finding of such a defect should institute a search for a loose body, and conversely, the finding of a loose body in a joint should evoke a search for its site of origin.

CASE REPORTS

The following cases are reported to illustrate the characteristic history and roentgen findings which occur in osteochondritis dissecans. In addition to the knee, cases involving sites of origin less frequently encountered are reported. These include the talus, the patella, the supratrochlear septum of the humerus, the proximal phalanx of the great toe, the glenoid of the scapula and the head of the radius.

The first case is typical of osteochondritis dissecans affecting the medial femoral condyle.

Case 1. A thirty-one year old soldier was admitted to the hospital with a history of injury to his right knee four years previously, when he dropped a 65 pound metal clutch on the knee. Since that time, he has had pain, swelling and "locking" of the knee. After entering the military service these symptoms were accentuated by marching and drilling. He found that frequently when he tried to rise from a kneeling position, he had to shake the knee to release a "locking." He entered a Station Hospital on August 13 because of pain and swelling of the knee. Traction for about two weeks failed to give relief.

On physical examination, the right knee showed a moderate diffuse swelling and marked limitation of motion. A freely movable body could be palpated over the medial aspect of the joint.

Roentgenographic examination on September 5 showed a fragmented osseous body measuring 2.5 by 1 by 0.5 cm. lying in the anteromesial portion of the joint space (Fig. 11). There was a large defect in the posterolateral aspect of the articular surface of the medial femoral condyle. This could be seen only in the "tunnel" view. Two small bony fragments were also present in the posterior joint space.

Fig. 10. Osteochondritis dissecans of the head of the radius, bilateral. Roentgenographically, this patient shows failure of epiphyseal closure in other bones. He is awaiting operation.

Arthrotomy was done on September 14. "A 3 inch medial parapatellar incision was made and the joint space opened. A smooth irregular cartilaginous free body was removed from the suprapatellar pouch. It measured about 3 by 2 by 1 cm. A healed area at the upper margin the medial femoral condyle, covered with fibrocartilage, was the site of the joint mouse. A pannus formation slightly overgrew the condylar lip in this area."

The pathologist reported "a piece of white cartilaginous tissue 2.7 by 2 by 0.6 cm. One

surface is smooth except for three shallow creases near one end; the other surface is pockmarked and fissured. Sectioning reveals an ossified center covered by cartilage 0.1 by 0.4 cm. in thickness. Microscopically the sections reveal that the superficial layer of tissue is composed of hyaline cartilage on one side and fibrocartilage on the other. Necrotic bone in the center. Diagnosis: osteochondritis dissecans."

The occurrence of osteochondritis dissecans in the talus is very infrequent[6,17].

There was no evidence of osteochondritis. On probing, an area of softening was detected on the anterosuperior medial aspect. A portion of degenerated subarticular bone about 0.5 cm. in diameter was easily removed with knife and curet."

Pathological Report. "I. A round nodule of hard, gray, smooth cartilaginous tissue measures 0.8 by 0.6 by 0.5 cm. On section, the surface shows a thick rim of pearly gray cartilage with a central core of yellow trabeculated bone.

FIG. 11. Case 1. Osteochondritis dissecans of the medial femoral condyle. The fragmented calcific body noted in the anteromesial aspect of the knee joint had moved into the suprapatellar pouch at the time of operation. On removal it measured 2.7 by 2 by 0.6 cm. A small defect which can be seen only in the "tunnel" view (arrow) was found to be the site of origin of the loose body. It is evident that the loose body must have grown since the time of its separation.

CASE II. A twenty-five year old Air Corps Cadet entered the hospital because of pain in his right ankle. He stated that he had twisted the ankle on November 11, when he stepped in a hole in the pavement during physical training exercises. Pain and disability had existed since that time. There had been no history of previous trauma.

Examination of the right ankle showed slight swelling, and moderate tenderness on palpation over the lateral aspect of the right foot below the ankle.

Roentgenographic examination on December 6 showed a smooth, oval, radiotranslucent area measuring 1 cm. in diameter involving the superior and medial aspect of the articular surface of the talus. Within this area was a smaller area of increased density due to the sequestrum (Fig. 12).

Operation was done on December 10. "The right ankle joint was opened. The superior aspect of the talus was exposed and examined. II. Several thin, rather soft, irregular fragments of cartilage which aggregate a mass measuring 0.8 cm. in diameter. Microscopic: I. Nodule composed of irregular thick trabeculae of bone, overlaid by a cap of hyaline cartilage. There is a sharp line of demarcation between cartilage and bone. The intertrabecular spaces are filled with loose, edematous connective tissue, which in some areas gradually becomes transformed into cartilage and then the cartilage into bone. II. Hyaline cartilage with areas of degeneration and irregular calcification of the matrix. Diagnosis: osteochondritis dissecans."

In this case, the articular cartilage had not become involved. At the time of operation the disease was confined to the subchondral bone. The difficulty frequently encountered by the surgeon in finding the lesion is well illustrated in this case. Only by careful probing was the margin of the sequestrum found.

We have found the patella involved in 4 cases. The 2 cases which were operated upon are reported in detail.

Case III. A twenty-one year old private was admitted on May 5, 1943, with a history of recurrent pain in the right knee since November, 1942. At that time, while going over the hurdles on an obstacle course, he caught his heel and bent the knee suddenly. He had sudden pain and felt a snap in his knee. The part was swollen for about three weeks and was intermittently painful until he entered the hospital. Several times, while walking, he felt something "pop" out of the lateral side of the knee, and he had to stop and push the piece in.

On physical examination hyperflexion caused pain. There was no evidence of swelling or instability.

Roentgen examination on May 6 showed a 1 by 0.25 cm. loose body in the anterior portion of the knee joint. There was a moderate-sized defect in the posteroinferior aspect of the patella with a small detached sequestrum in this region (Fig. 8*a*).

At operation on May 13 "the joint was inspected and two loose bodies were removed. The patella was inspected and on its under surface there was a defect partially covered with cartilage."

Pathological Report. "Gross: I. Several small, soft, purplish-pink fragments of tissue measuring 1.0 cm. in diameter. II. Two pearly-gray, oval, hard, smooth pieces of tissue measuring 1.9 by 0.4 and 0.9 by 0.7 by 0.3 cm. respectively. Microscopic: I. Multiple, small fragments are partly and some are completely covered with fibrinoid material. II. Oval nodules with a central core of coarse, irregular, degenerating bone trabeculae. This central nucleus is covered with a thick layer of hyaline cartilage, with fibrous tissue peripherally, in which small fragments of necrotic bone are visible. Diagnosis: osteochondritis dissecans."

Fig. 12. Case II. Osteochondritis dissecans in the talus (arrow). In the oblique projection the lesion is also clearly evident (arrow).

Case IV. A twenty-four year old soldier entered the hospital on June 15, 1944, stating that ever since he could remember, his left knee had "gone out of joint" at intervals. There was no history of a precipitating injury. Since his induction into the Army twenty-one months before, he had had six episodes of the knee "going out of joint."

On physical examination the left knee showed no palpable deformity, abnormality or demonstrable swelling. There was acute tenderness over the medial aspect of the joint. Extension of the knee caused pain.

On roentgenographic examination on June 16, 1944, there was a small loose fragment in the anterior aspect of the left knee joint at the level of the inferior margin of the patella. Another bony fragment was slightly separated from the lower margin of the patella, but was not displaced. The inferior aspect of the patella showed a small defect (Fig. 8*b*).

At operation on June 30 "a median para-

patellar incision was made nearer to the patella than is usual. The left knee joint was exposed. A bean-sized, smooth, glistening body was seen lying at the anterior attachment of the anterior cruciate ligament. Inspection of the femoral condyles showed normal cartilaginous surfaces. The under surface of the patella was rough, shaggy and fibrillated, and apparently had undergone extensive osteochondritic changes. A small pea-sized body was palpable at the lower pole of the patella, within the patellar tendon. This was not removed."

Pathological Report. "I. Approximately 20 irregular pieces of pearly-gray fibrocartilage. II. A smooth egg-shaped white hard nodule measuring 0.9 by 0.6 by 0.4 cm. On section, irregular yellowish lines were found along the margin, suggesting fine bone lamellae. Microscopic: Several pieces of mature hyaline and a section of fibrocartilage are not remarkable. In addition an oval nodule is present which shows hyaline cartilage on one surface and fragments of necrotic bone on the other. Diagnosis: osteochondritis dissecans."

The patella is an infrequent site of osteochondritis dissecans. The bone defect and the loose fragment are often small and may be overlooked. In the second case, the second loose fragment is undoubtedly due to a congenital anomaly and appears in otherwise normal knees on rare occasions.

The following case is one of osteochondritis dissecans occurring in the distal end of the humerus, arising from the supratrochlear septum:

CASE V. A twenty-four year old soldier entered the hospital complaining of intermittent pain in the right elbow for four years. At that time, while playing football, he injured his right elbow and had difficulty in straightening the arm. Roentgenograms taken at the time of injury were said to show no evidence of fracture. Heavy work caused pain and he was obliged to hold the arm at his side. There was no history of locking but extension of the forearm was limited.

On examination, the patient held his right elbow close to the body with the forearm in flexion. He could not raise his arm over his head and was unable to fully extend his forearm.

Roentgenographic examination on July 14, 1944, showed a large dense button of bone measuring 2 cm. in diameter arising from a depression in the posterior aspect of the lower end of the humerus (the supratrochlear septum). Another smaller osseous fragment arose anteriorly at the same level. This measured 1 cm. in diameter (Fig. 9). The roentgen diagnosis was osteochrondritis dissecans.

Because of the disability and because the condition had existed prior to induction into the Army a month before, the patient was honorably discharged.

The appearance of the lesion is characteristic of osteochondritis dissecans seen elsewhere. Multiple foci were present; one large sequestrum posteriorly and at least one anteriorly.

DIFFERENTIAL DIAGNOSIS

Osteochondritis dissecans must be differentiated roentgenologically from those conditions in which loose bodies appear in the joint space, and clinically, from those conditions which are accompanied by pain, swelling and "locking" of a joint.

Osteochondritis dissecans must be differentiated mainly from (1) traumatic loose bodies; (2) osteochondromatosis; (3) detached osteophytes; (4) neuroarthropathies, and (5) tuberculosis.

Traumatic Loose Bodies. Loose bodies in the joint space may occasionally result from a fracture with displacement of a small bony fragment. Milgram[18] has reported cases of tangential osteochondral fracture of the patella which may simulate osteochondritis dissecans. In these cases there is a history of acute trauma to the knee with tearing of a small fragment from the medial and inferior aspect of the patella. Early roentgenograms may serve to demonstrate the recent fractures. If operation is performed early, a fresh fragment of bone and cartilage may be found loose in the joint, floating in sanguineous fluid containing clots. Fractures of the femoral condyles may also be observed at this time. Microscopically, the cartilage is normal. The presence of blood, the associated fractures, and the viable bone fragment serve to differentiate this condition if the operation is performed early. With delay, there is a recession of the hemarthrosis and a degeneration of the separated fragments, with the

result that the condition may become indistinguishable from osteochondritis dissecans.

Osteochondromatosis. In synovial osteochondromatosis, which is the result of hypertrophy of the synovial villi, or possibly benign tumors of the synovial membrane, loose bodies appear, usually in the knee, but occasionally in the shoulder or hip joints. These are multiple, as many as a hundred or more may fill and distend the joint space. The bodies are disc-like, dense at the center and periphery, and have mottled calcification in between. The great number of the loose bodies, the mottled calcification of the fragments and the absence of a bony defect distinguishes this condition from osteochondritis dissecans.

Detached Osteophytes. Occasionally osteophytes become detached from the margins of joints in hypertrophic or degenerative arthritis. These produce locking of the joint similar to other joint "mice." The much older age group of these patients, the polyarticular involvement of the arthritic changes, the roentgenologic evidence of irregular eburnation of the articular surfaces of the bones accompanied by marginal lipping or spur formation, and the absence of the characteristic bony defect help to differentiate this from osteochondritis dissecans.

Neuroarthropathies. In joints affected by dissolution of the peripheral sensory innervation, changes result which may produce loose bodies. This is seen most commonly in syringomyelia, tabes dorsalis and leprosy. In the arthropathies accompanying these diseases, as a result of the loss of sensation and repeated trauma, there is eburnation, osteophyte formation and fragmentation of the articular cortices, with the subsequent formation of loose bodies, and narrowing of the joint space. The occurrence of these conditions in an older age group, the history and neurologic findings, as well as the roentgenologic appearance, are distinguishing features even in early cases.

Clinically, a ruptured meniscus may produce a typical history of "locking" of a joint such as is seen in osteochondritis dissecans. In this condition, however, there is usually a history of sudden twisting with the knee fixed. The characteristic roentgen appearance of osteochondritis dissecans is absent

TREATMENT

The treatment of osteochondritis dissecans may be either conservative or surgical. In cases without symptoms in which the sequestrum is not detached, it is generally agreed that no treatment is indicated. Complete healing may occasionally occur with complete disappearance of the necrotic focus.[16] Recently, Strange[25] reported a case in a four and a half year old child in whom immobilization of the part resulted in a return to normal.

In those patients having osteochondritis dissecans without separation but in whom symptoms are present, physiotherapy has been tried but without definite improvement. These are probably best treated by operation.

When there are loose bodies in the joint it is generally agreed that surgical removal is the only acceptable method of treatment. King[13] reports 88 per cent satisfactory results in his series treated surgically. When the symptoms are of long duration, with several loose bodies, results are not as satisfactory inasmuch as there is usually an accompanying arthritis or synovitis. The fact that the arthritis and synovitis may develop as a result of loose bodies is a definite indication for surgery, even in those cases in which the symptoms may be mild.

SUMMARY

Osteochondritis dissecans is a non-infectious lesion in which there is necrosis of a segment of subchondral bone with formation of an osseocartilaginous sequestrum. Trauma, either direct or indirect, probably plays an important rôle in the formation of this disease. Since no early cases have been observed, its exact mode of origin is undetermined. The roentgeno-

graphic appearance is characteristic; there is a sharply defined cavity from which arises a sequestrum of bone. Depending upon the age of the lesion, the sequestrum may still be within the cavity and covered by unbroken cartilage or may have been extruded into the joint space. This condition occurs more frequently than is currently accepted. Among 891 knees examined in a two year period, exclusive of cases having gunshot wounds or obvious fractures, thirty-three cases of osteochondritis dissecans were found. Of these, twenty-seven involved the medial femoral condyle, two the lateral femoral condyle, and four the patella. A significant history of trauma was present in twenty-two of these cases. Of these cases, nineteen of the medial femoral condyle and one of the lateral femoral condyle were proved by operation. Two of the cases involving the patella were also operated upon. We have seen, during the same interval, one proved case occurring in the talus. Also observed during this time were three cases involving the supratrochlear septum of the humerus, one case in the glenoid of the scapula, two cases with bilateral involvement of the head of the radius and one case in the proximal phalanx of the great toe. While these were not proved by operation, the characteristic roentgenographic criteria for a diagnosis were fulfilled.

The differential diagnosis is discussed and several cases, some of which are in unusual locations, are reported in detail and illustrated.

Box 944
Haverhill, Mass.

REFERENCES

1. ALLAN, J. H., and Nicholson, J. T. Knee injuries in service personnel. *U. S. Nav. Med. Bull.*, 1944, *43*, 63–72.
2. AXHAUSEN, G. Quoted by Phemister.[22]
3. BALENSWEIG, I. Osteochondritis dissecans of the knee. *J. Bone & Joint Surg.*, 1925, *7*, 465–468.
4. BERNSTEIN, M. A. Osteochondritis dissecans. *J. Bone & Joint Surg.*, 1925, *7*, 319–329.
5. CARRELL, B., and CHILDRESS, H. M. Osteochondritis dissecans of the metatarsal head. *J. Bone & Joint Surg.*, 1940, *22*, 442–443.
6. COBEY, M. C. Osteochondritis dissecans of the astragalus. *Mil. Surgeon*, 1943, *93*, 184–186.
7. CONWAY, F. M. Osteochondritis dissecans. *Am. J. Surg.*, 1937, *38*, 691–699.
8. FAIRBANK, H. A. T. Osteo-chondritis dissecans. *Brit. J. Surg.*, 1933, *21*, 67–82.
9. FREIBERG, A. H. Osteochondritis dissecans. *J. Bone & Joint Surg.*, 1923, *5*, 3–17.
10. HERTZ, J. Quoted by Hutchison.[11]
11. HUTCHISON, R. G. Osteochondritis dissecans. *Brit. J. Radiol.*, 1943, *16*, 147–149.
12. KILFOY, E. J. Osteochondritis dessecans of the left elbow. *Am. J. Surg.*, 1941, *53*, 496–499.
13. KING, D. Osteochondritis dissecans. *J. Bone & Joint Surg.*, 1932, *14*, 535–544.
14. KING, D., and RICHARDS, V. Osteochondritis dissecans of the hip. *J. Bone & Joint Surg.*, 1940, *22*, 327–348.
15. KÖNIG, F. Quoted by Richards.[23]
16. LIEBMAN, C., and ISEMAN, R. M. Osteochondritis dissecans. AM. J. ROENTGENOL. & RAD. THERAPY, 1940, *43*, 865–870.
17. MENSOR, M. C., and MELODY, G. F. Osteochondritis dissecans of the ankle joint; use of tomography as a diagnostic aid. *J. Bone & Joint Surg.*, 1941, *23*, 903–909.
18. MILGRAM, J. E. Tangential osteochondral fracture of the patella. *J. Bone & Joint Surg.*, 1943, *25*, 271–280.
19. MORTON, H. S., and CRYSLER, W. E. Osteochondritis dissecans of the supratrochlear septum. *J. Bone & Joint Surg.*, 1945, *27*, 12–24.
20. MUNRO, A. Quoted by Phemister.[22]
21. MURPHY, F. G. Osteochondritis dissecans of the elbow joint. *J. Bone & Joint Surg.*, 1939, *21*, 464–466.
22. PHEMISTER, D. B. Causes of and changes in loose bodies arising from the articular surface of the joint. *J. Bone & Joint Surg.*, 1924, *6*, 278–315.
23. RICHARDS, G. E. Osteochondritis dissecans. AM. J. ROENTGENOL. & RAD. THERAPY, 1928, *19*, 278–284.
24. ROMBOLD, C. Osteochondritis dissecans of the patella. *J. Bone & Joint Surg.*, 1936, *18*, 230–231.
25. STRANGE, T. B. Osteochondritis dissecans. *Am. J. Surg.*, 1944, *63*, 144–145.
26. STEIN, G. H., IKINS, R. G., and LOWRY, F. C. Osteochondritis dissecans. *Am. J. Surg.*, 1944, *64*, 328–337.
27. WAGONER, G., and COHN, B. N. E. Osteochondritis dissecans. *Arch. Surg.*, 1931, *23*, 1–25.

Vol. 57, No. 1

FRACTURES OF THE FEMORAL NECK FOLLOWING PELVIC IRRADIATION*

By WENDELL P. STAMPFLI, M.D.†
DENVER, COLORADO
and
H. DABNEY KERR, M.D.
IOWA CITY, IOWA

THE object of this paper is to present our experience with 12 patients having fracture of the neck of the femur secondary to pelvic irradiation. The pathology, diagnosis and treatment of these injuries will be discussed. Although no autopsy material is available, several biopsies were taken. Our findings, as well as other evidence in the literature, has led to a newer concept regarding the etiology of these fractures. Contrary to the views expressed in some contributions on this subject, we think that the risk of serious skeletal damage during extensive irradiation for pelvic neoplasm is not great. The incidence is not sufficient to justify the omission of lateral fields, and the condition is not serious enough when it does occur to deter us from our efforts to control pelvic carcinoma.

HISTORICAL

It was only a few weeks after the discovery of roentgen rays that their biologic effects were first observed. Soft tissue reactions occupied the attention of the earliest investigators but not until the advent of intensive radiation therapy for neoplastic disease was skeletal damage considered a problem. Much good work has been done and something is now known about the changes produced in bone by irradiation, but the manner in which these changes are brought about is not fully understood. Clinically, it has been found that heavy irradiation lowers the powers of resistance in bone and predisposes to further damage by infection and trauma. Regaud,[29] in 1922, made this observation and referred to cases that had developed necrosis of the maxillary bones following the treatment of intra-oral carcinoma. In 1926, Ewing[10] reported that normal bone is susceptible to heavy irradiation, and that large doses incite a chronic productive radiation "osteitis." In this condition, the bone becomes hard and brittle and spontaneous fractures often occur. The periosteum becomes thickened and hyalinized, bits of necrotic bone appear, and the shaft may be greatly increased in thickness. In another communication,[11] he suggested that the dead bone may act like transplanted bone and be gradually replaced by slowly growing new bone.

The most common skeletal injury now encountered secondary to irradiation is fracture of the femoral neck. Baensch[2] recorded the first case in 1927. His patient suffered bilateral spontaneous fractures following irradiation for carcinoma of the cervix. He believed that necrosis secondary to vascular damage was the predisposing factor. In 1932,[3] he added a second case and during the same year Philipp[27] reported 4 cases. He believed that lateral fields contributed to the incidence of necrosis and advised smaller doses through those fields. Kropp[18] mentioned a case in 1934 and Baclesse and Regaud[1] have observed 3 cases.

In 1936, for the first time in this country, Dalby, Jacox and Miller,[9] from the University of Michigan, presented a study of 14 cases of irradiation fracture of the femoral neck. It is worthy of note that none of their patients received treatment through lateral fields. Miller and Folsome,[19] from the same institution, added 21 more

* From the Department of Radiology, University Hospitals, State University of Iowa, Iowa City, Iowa.
† Saint Luke's Hospital, Denver, Colorado.

cases in 1938. Peck[21] gave an account of 28 cases in 1939. He included the original 14 presented in 1936, by Dalby, Jacox and Miller and some of those mentioned by Miller and Folsome. Kalayjian,[17] Okrainetz and Biller,[20] Conzett,[7] Hight,[15] Baker,[4] and Barden[5] have each reported single cases. Healy and Frazell[14] reported 2 cases in 1937. A censorious investigation of osseous irradiation injuries was made by Strauss and McGoldrick[32] in 1941, and they reported 4 more cases of irradiation fracture of the femoral neck. They believed that the depth dose administered to the lesion through lateral fields was not enought to justify the risk of osseous damage. Slaughter,[31] in 1942, contributed an excellent study of osteitis and fractures following irradiation. He reported 7 more cases with fracture of the femoral neck and concluded that bone is one of the more radiosensitive tissues of the body. He believed that the incidence of such fractures secondary to irradiation was too small to be of any real significance, and suggested that they might be eliminated by shielding the femoral neck. During the same year Truelsen[33] presented 3 more cases and drew interesting conclusions regarding the effect of irradiation on bone. Sections from his cases showed no particular vascular damage but did show a remarkable lack of cellular reactions; there were no osteoblasts and few osteoclasts in the sections. During a discussion at the meeting of the American Radium Society in June, 1938, Johnston, Schmitz and Costolow[16] each mentioned single cases, but gave no details. Gratzek, Holmstrom and Rigler,[13] in a more recent report, describe extensive damage to the pelvis and upper femurs following pelvic irradiation. They add 12 more fractures of the femoral necks. The prognosis in these injuries, they believe, is quite good, since recovery occurs in many instances with relatively little disability. Peck,[12] in a personal communication, stated that he had seen 3 more cases since his original report in 1939. He believes that lateral fields are not an important contributing factor since most of the cases he has seen did not receive treatment through them.

PATHOLOGY OF IRRADIATION FRACTURES

General Considerations. Traumatic fracture of the femoral neck usually occurs in elderly persons. It is two to three times more common in women than in men and for some unexplained reason is found more often on the left. It may result from a trivial strain or torsion from indirect violence, and if the victim falls, the fall is more apt to be the result, rather than the cause, of the fracture. The most common predisposing factor is osteoporosis incident to old age. Demineralization weakens the bone and there is a general loss of elasticity so that the bone is more brittle. In old persons, reflexes are less active, and sudden unusual strains may be thrown upon the skeleton. The femoral necks, because of their position are especially susceptible to such stresses, which sometimes cause fractures.

Pathologic fractures are rare but may occur secondary to neoplasm, senile osteoporosis, radiation changes and metabolic and endocrine abnormalities. Post-irradiation changes are becoming increasingly important.

Anatomic and Physical Considerations. The structure of bone presents certain characteristics that should be emphasized in a study of osseous irradiation injuries. It was Ewing[11] who first pointed out that the intricate network of vascular channels and the fine system of lacunae might be a very marvelous, but at the same time a very hazardous, arrangement. He believed that the blood supply was peculiarly liable to strangulation from occlusion of the vessels in the haversian and Volkmann's canals. The bone cells lying in lacunae with their filaments passing through minute canaliculi are intimately related to the dense, earthy, intercellular matrix. The latter contains considerable calcium and phosphorus and is believed to produce more secondary radiation than is produced by soft tissue.

Not much is known about the absorption

characteristics of bone and the nature of its secondary radiation. Failla[12] has shown that bone absorbs appreciably more radiation than do the soft tissues and, according to a personal communication from him quoted by Slaughter,[31] this may amount to as much as 30 to 40 per cent. Numerous investigators attempting to explain the susceptibility of bone to radiation have claimed that the calcium and phosphorus may be responsible for more intense and highly destructive secondary rays. This has never been proved experimentally, however. Quimby[28] calls attention to the fact that the only source of these secondary rays is the energy absorbed from the primary beam. Since, in bone, this is no more than twice as much as is absorbed in soft tissue, it is obvious that the intensity of this secondary beam cannot be as great as many authors have indicated. Excepting scatter, the hardest component of this secondary beam is the characteristic radiation from calcium, which is very soft.

Lining the periosteum, the medullary cavity and the haversian canals are the osteoblasts. It might be well for us to investigate the radiosensitivity of these cells. Their destruction might be brought about by ordinary doses of radiation, and their death might contribute to the changes leading to fractures and slow repair.

Radiation Osteitis. Osteoporosis is one of the more conspicuous microscopic findings and is extremely significant from the standpoint of irradiation fracture. Boyd[6] states that the two chief factors in the process of bony absorption are vascular absorption and osteoclastic activity. Vascular absorption plays the more important rôle and is brought about by the persistent dilatation of the vessels in the haversian and Volkmann's canals. Hyperemia is followed by widening of the canals at the expense of bone, and if absorption becomes very marked, rarefaction may result. Osteoclasts probably play a secondary part in the process and it is probable that decalcification must first occur before the osteoclast can exert its phagocytic action. Decalcification is a little understood physiochemical phenomenon brought about by the tissue juices, or possibly by the indirect effect of the osteoclasts themselves. Actual death of the bone may ensue as a direct effect of irradiation on bone cells, as well as from vascular damage. Dahl[8] was able to demonstrate that osteoblasts were more sensitive to radiation than osteoclasts and he suggested that doses just large enough to inhibit the former may not affect the latter. Zöllner[34] believed that the osteoporosis following irradiation may be the result of a disturbance in the normal balance between resorption and production of bone on account of the sensitivity of osteoblasts.

In a personal communication, Phemister[23] stated that he had an opportunity to study several cases with post-irradiation fractures in the femoral necks. He believes that both the bone and the soft parts of the femur are damaged, but not to the point of complete necrosis. He has seen osteoporosis, fibrosis and in some cases endarteritis. In other cases, no definite vascular narrowing and thickening was noted. Osteoclastic activity was observed and he suspects that scavenger cells might be more active than osteoblasts.

Biopsies were obtained from 3 of our patients (P. P., H. O., and J. C.) at the time osteotomies were performed. The surgeons, in 2 of these cases, were impressed by the thin, fragile, "shell-like" qualities of the bone. By far the most outstanding feature of the microscopic sections was osteoporosis. The trabeculae were thin and bone cells were scarce. In 2 cases, there were no osteoblasts and very few osteoclasts. In the other case, the fracture was a year old, and osteoblastic as well as osteoclastic activity was seen. Although a few spicules of dead bone were evident, most of the examined tissue was viable. None of these showed vascular narrowing and the degree of thickening was compatible with the patient's age.

In view of our findings, as well as those reported by others, it seems probable that

TABLE I

Patient	Age	Weight When Treated lb.	Diagnosis	Fields		Size of Fields in cm.	Air Dose in Roentgens to Each Field	Total Air Dose in Roentgens	Tumor Dose in Roentgens	Length of Course in Days	Hip Involved	Interval in Months between Treatment and Symptoms	Interval in Months between Treatment and Roentgen Proof of Fracture
J. C.	62	142	Carcinoma of cervix	1st course	1 anterior	20×20	700	15,500	3570	33	Right	None	32
					1 posterior	20×20	600						
				2nd course	2 anterior	10×15	1700				Left	31	32
					2 posterior	10×15	1800						
					2 lateral	10×15	1800						
					2 gluteal	10×15	1800						
H. O.	37	90	Carcinoma left ovary	1st course	2 anterior	10×15	2000	16,800	3660 right ovary	30	Right	32	32
					2 lateral	10×15	2000						
					2 posterior	10×15	2000						
				2nd course	1 left ant.	8×10	1600		6320 left ovary	12 more	Left	13	29
					1 left post.	8×10	1600						
					1 left lat.	8×10	1600						
C. E.	58	161	Carcinoma of cervix		2 anterior	10×15	2100	17,000	3392	30	Right	8	15
					2 posterior	10×15	2100						
					2 lateral	10×15	2100						
					2 gluteal	10×10	2100				Left	27	37
					1 perineal	8×10	800						
H. B.	59	148	Carcinoma of bladder		2 anterior	10×15	1800	15,200	3527	47	Left	25	29
					2 posterior	10×15	1800						
					2 lateral	10×15	1800						
					2 gluteal	10×15	1800						
					1 perineal	8×10	800						
N. S.	61	174	Carcinoma of cervix		2 anterior	10×15	2100	15,800	2917	33	Right	27	39
					2 posterior	10×15	1800						
					2 lateral	10×15	1800						
					2 gluteal	10×15	1800						
					1 perineal	8×10	800						
A. T.	66	116	Carcinoma of cervix		2 posterior	10×15	2100	11,400	2480	25	Left	7	10
					2 anterior	10×15	1800						
					2 lateral	10×15	1800						
L. M.	57	170	Carcinoma of cervix		2 anterior	10×15	2100	21,600	3790	49	Left	29	34
					2 posterior	10×15	2100						
					2 lateral	10×15	2100						
					2 gluteal	10×15	2100						
					2 ant. obl.	10×15	1000						
					2 post. obl.	10×15	1000						
					1 perineal	8×10	800						
B. P.	34	120	Carcinoma of cervix		2 anterior	10×15	1800	15,400	2845	39	Right	40	43
					2 posterior	10×15	1800						
					2 lateral	10×15	1800						
					2 gluteal	10×15	1800						
					1 perineal	8×10	1000						
H. S.	55	140	Carcinoma of cervix		2 anterior	10×15	2000	16,800	3746	42	Right	22	24
					2 posterior	10×15	2000						
					2 lateral	10×15	2000						
					2 gluteal	10×15	2000				Left	30	30
					1 perineal	8×10	800						
P. P.	55	118	Carcinoma of cervix		2 anterior	10×15	2200	13,200	2600	34	Right	12	17
					2 posterior	10×15	2200						
					2 lateral	10×15	2200						
J. N.	65	120	Carcinoma of cervix		2 anterior	10×15	1800	13,200	2800	51	Left	26	32
					2 posterior	10×15	1400						
					2 lateral	10×15	1600						
					2 gluteal	10×15	1700						
					1 perineal	8×10	200						
A. K.	63	192	Carcinoma of cervix		2 anterior	10×15	2400	22,200	3040	49	Right	32	34
					2 posterior	10×15	2400						
					2 lateral	10×15	2400						
					2 ant. obl.	10×15	1600						
					2 post. obl.	10×15	1600						
					1 perineal	8×15	1400						

the most important change following intensive irradiation of the femoral neck is osteoporosis. Osteoporosis may result from vascular absorption and osteoclastic activity while normal recovery is impeded by the death of osteoblasts. Necrosis secondary to vascular occlusion probably does not occur in the femoral neck as often as it does in the mandible which may receive extremely high doses during the treatment of intra-oral neoplasms. If vascular damage were the predisposing cause of the fractures, it seems likely that more aseptic necrosis of the femoral head would occur. Actually this complication is not common following fractures due to irradiation. Phemister[23] saw vascular damage in some cases, but it was entirely absent in others. Truelsen[33] examined sections from the femoral neck of one of his patients who had suffered a fracture following intensive irradiation. He saw no particular changes in the blood vessels. Our cases also fail to show such changes.

Phemister[24,25,26] has described the process of repair in aseptic necrosis. He has observed repair following necrosis from radium, as well as from other causes, and has explained how, after bone dies, it is slowly resorbed and later replaced by a process of "creeping substitution" from adjacent viable bone. The reparative process following irradiation osteitis is generally retarded, however, because of the extensive damage done to the surrounding tissues including the vessels, bone cells, and periosteum. Its progress is slow and because of the lowered resistance, the bone is highly susceptible to further damage from infection and trauma.

ANALYSIS OF CASES

Approximately 1,372 patients have received intensive pelvic irradiation in this hospital during the years between January 1, 1931 and July 1, 1943. Most have been followed, and only 14 cases with fracture of the femoral neck have been discovered. Two of these gave a history of trauma and for other reasons were not included in this study. The 12 cases presented here were treated externally about the pelvis with 200 kv. 50 cm. distance, and Thoraeus filter (0.4 mm. Sn, 0.25 mm. Cu, 1.0 mm. Al) which produced a beam having a half-value layer of 1.95 mm. Cu. The dosage and method of treatment are indicated in Table 1. Ten of these patients had carcinoma of the cervix while the other 2 had carcinoma of the ovary and bladder, respectively. In each case, the primary diagnosis was histopathologically confirmed. Radium was applied directly to the cervix in 9 cases and the dose was 1,500 milligram-hours in one case and ranged from 5,000 to 6,050 mg-hr. in others. The small amount reaching the femurs from this source was considered negligible.

These 12 patients with irradiation damage to the femoral necks constitute an incidence of only 0.87 per cent. Dalby, Jacox and Miller reported an incidence of 2.1 per cent. Our youngest patient was thirty-four years old at the time of fracture and the oldest sixty-six; the average age was fifty-six. None of our cases showed the extensive pelvic damage reported by Gratzek, Holmstrom and Rigler,[13] but 4 of them had bilateral fractures while 1 showed sclerotic post-irradiation changes in the opposite hip without fracture. Four cases showed roentgen evidence of irradiation change in the pubic bones and 1 of these developed a fracture in the inferior ramus.

The average interval between the beginning of treatment and the first symptom referable to the affected hip was twenty-four months. Roentgenologic evidence appeared five months later. The shortest interval between treatment and roentgen proof of fracture was only ten months while the longest was forty-three months.

All of our patients received treatment through lateral fields and an appreciable tumor dose was delivered through them. The average depth dose on each side of the pelvis, 5 cm. from the midline in 10 cases of carcinoma of the cervix and 1 case of carcinoma of the bladder, was 3,155 r, and the average amount contributed by lateral fields was 800 r, or about 25 per cent.

We attempted to measure the amount of

radiation delivered to the femoral necks, but found that this could not be done accurately. Roentgenograms were made by arranging the patient and the therapeutic apparatus in the positions used for treatments. The film was inserted beneath the part to be studied and an appropriate exposure made. The femoral necks were within the direct beam of lateral fields, but only partially or not at all covered by anterior and posterior fields. This variation was due to the difference in the size of the patients, and made measurements of depth doses at the femoral necks unreliable.

TABLE II

Patient	Treatment	Interval	Result
	Group I. Incomplete fracture with subcapital sclerosis, impaction, or coxa vara, without a definite fracture line		
J. C. (rt)	Bed rest and crutches	4 mo.	Healed spontaneously
H. O. (rt)	Bed rest and crutches	62 mo.	Healed spontaneously
C. E. (rt)	None	24 mo.	Healed spontaneously
	Group II. Complete fracture with definite fracture line and little or no displacement		
H. B.	Threaded Steinmann pins	16 mo.	Firm union. Walks unaided but carries cane for security
N. S.	None, except shoe lift for shortening	7 mo.	Healed spontaneously, firm union; walks unaided
A. T.	None	12 mo.	Died one year after fracture; cause unknown. No information obtained
L. M.	Walking exercises, heat and massage	6 mo.	Healed slowly. Walks with cane
	Group III. Complete fracture with considerable displacement or with aseptic necrosis of the head		
B. P.	Subtrochanteric osteotomy	10 mo.	Firm osseous union at osteotomy site. Walks unaided
H. S. (rt)	Subtrochanteric osteotomy	17 mo.	Can walk unaided, but uses crutches for security
H. S. (lt)	Subtrochanteric osteotomy	14 mo.	Satisfactorily functioning osteotomies. Good healing
H. O. (lt)	Lorenz osteotomy	53 mo.	Healed with good function
P. P.	Lorenz osteotomy	49 mo.	Firm union. Walks unaided
J. C. (lt)	Smith-Petersen nail	4 mo.	Non-union. Nail removed. Died from pulmonary metastases
C. E. (lt)	Crutches—nothing else	11 mo.	Union taking place. Not yet complete
J. N.	None		Not followed
A. K.	Surgery contraindicated by cardiac complaint	26 mo.	Non-union

DIAGNOSIS

History. The average age of patients suffering from fracture of the femoral neck secondary to irradiation is appreciably lower than has been reported in studies made of similar fractures from all causes. Reggio and Wilson[30] found the average age of the latter group was sixty-three and a half years. Others report sixy-five. Our 12 patients had an average age of fifty-six years which compares well with reported figures for this type of fracture.

A few persons with post-irradiation fractures of the femoral neck give a history of mild trauma such as tripping on a rug, or twisting the leg. This was the case in 3 of our patients in whom the diagnosis of pathologic fracture was more difficult to confirm. The presence of characteristic

roentgen evidence of irradiation osteitis in the fractured bone or in the other bones of the pelvis will aid in making a differential diagnosis. Nine, or 75 per cent, of our patients gave no history of trauma. Three of them were bedfast at the time the fracture occurred. Two of these were recuperating from osteotomies on one hip and fracture of the opposite hip became apparent only when the patient got out of bed for the first time. The other patient was in bed with influenza when the fracture occurred.

In most cases, the first symptom is mild pain in the hip beginning one or two years after irradiation. The pain is at first continuous and dull but gradually becomes more severe and is often marked by sharp pangs on motion or weight bearing. The pain may be especially aggravated by rising from a sitting to a standing position. It may radiate, extending from the hip down the anterior aspect of the thigh to the knee. Many patients continue to walk, but most of them find that the affected hip is unstable and unable to bear their weight. Limping is a constant sign and is usually the result of pain, but may result from shortening with or without pain.

Physical examination reveals the usual signs of fractured hip. There may be shortening, external rotation, limitation of motion and crepitation. On the other hand, if the fracture is incomplete or impacted, these signs will not be manifest.

Fig. 1. August 18, 1944.

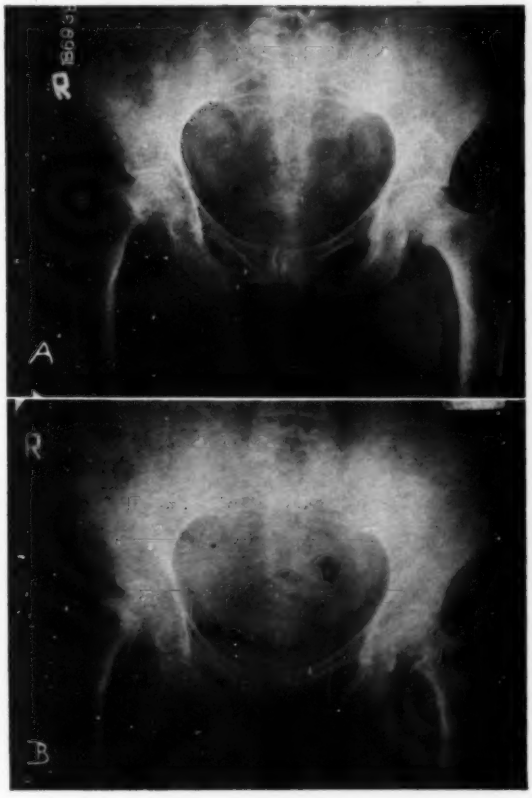


Fig. 2. N. S. *A*, November 1, 1944; *B*, May 31, 1945.

Roentgen Appearance. While pain may precede the fracture by several months, it is often possible at the time symptoms first appear to demonstrate early post-irradiation changes on roentgenograms. In the femoral neck, these changes appear as a subcapital zone of mottling. This narrow zone is often quite dense, with small irregular patches of rarefaction (Fig. 2*A*, left, Fig. 3*A* and *B*, right). Sometimes an incomplete incipient fracture can be made out and a zone of sclerosis may extend across the neck just distal to the head (Fig. 1; Fig. 5*A*). A complete fracture with separation of the fragments may never occur, but the zone may become more dense with thickening of the neck and varying degrees of coxa vara (Fig. 2*A*, right). Oc-

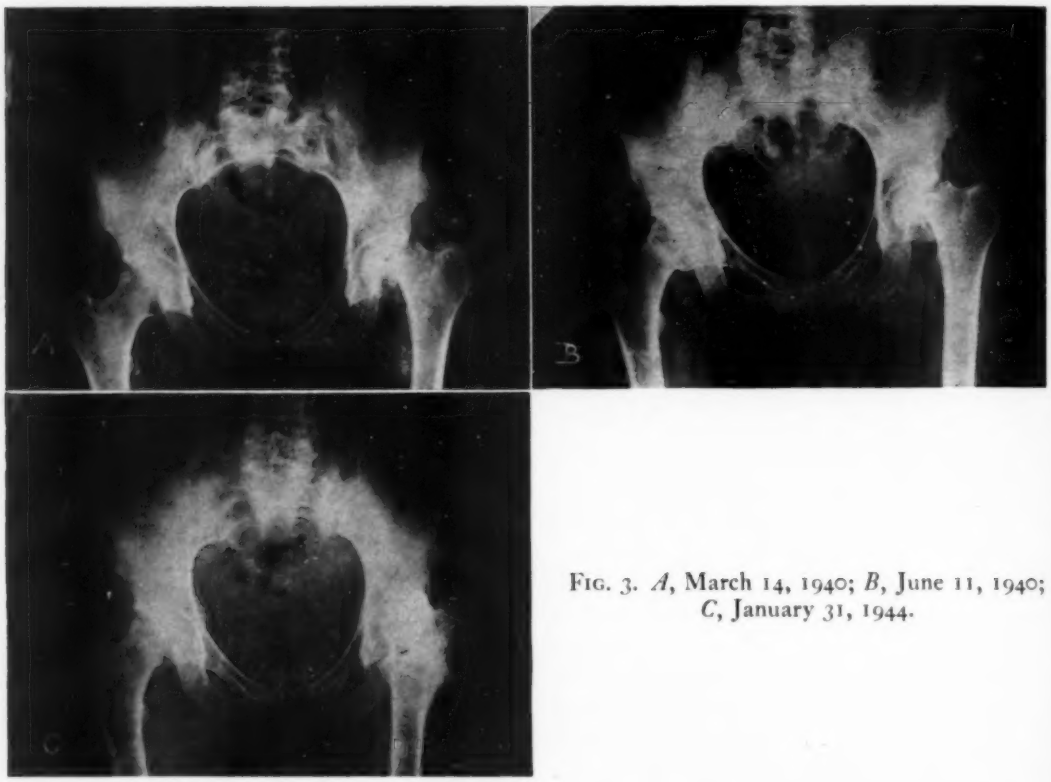


FIG. 3. *A*, March 14, 1940; *B*, June 11, 1940; *C*, January 31, 1944.

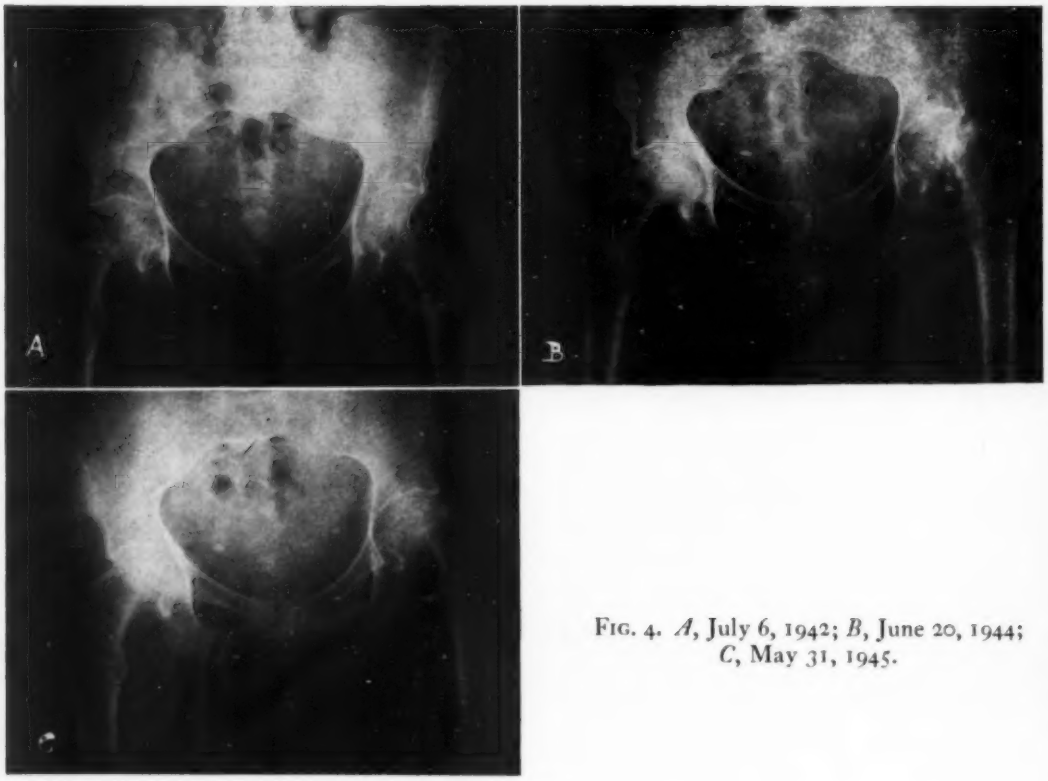


FIG. 4. *A*, July 6, 1942; *B*, June 20, 1944; *C*, May 31, 1945.

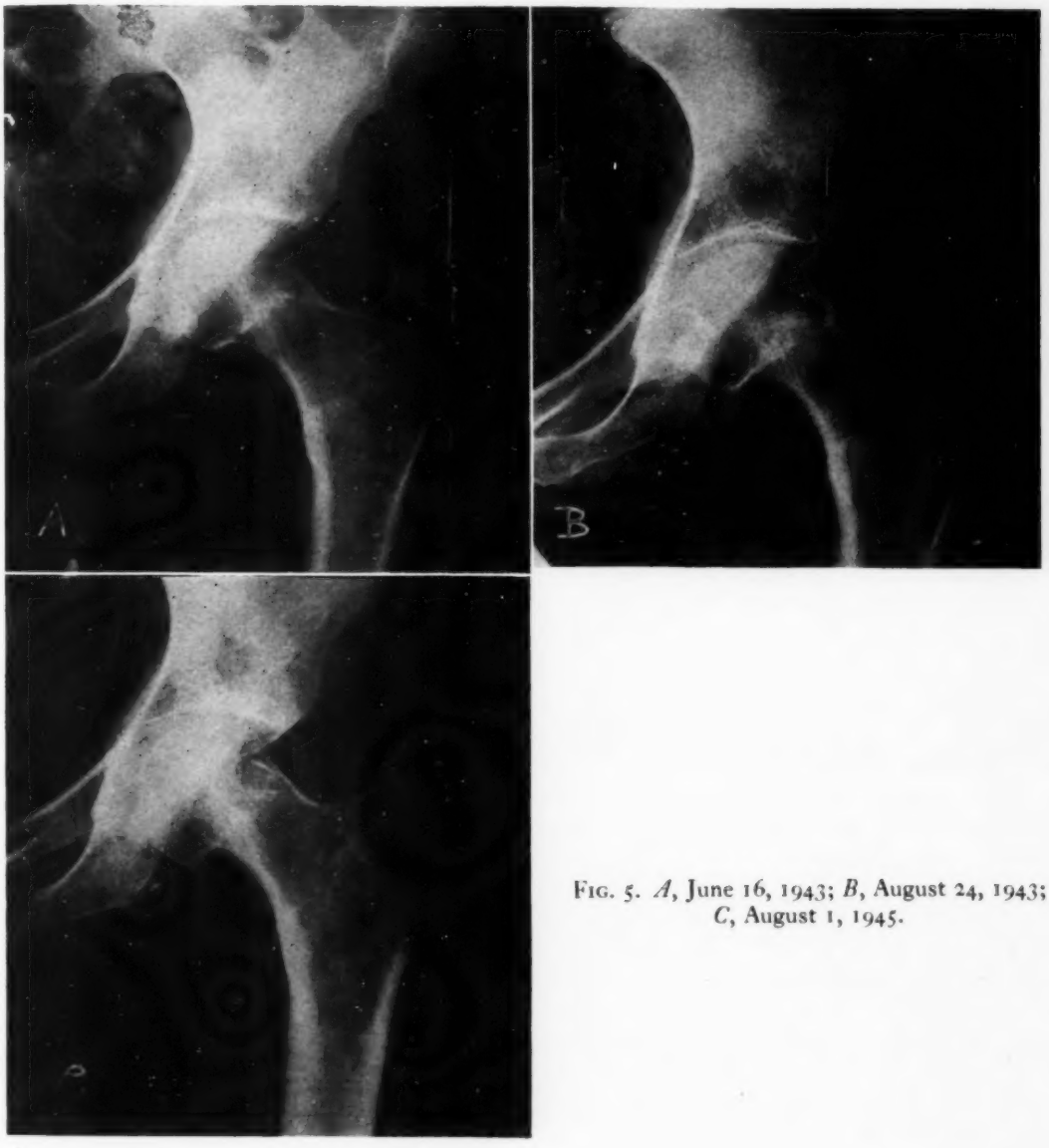


Fig. 5. *A*, June 16, 1943; *B*, August 24, 1943; *C*, August 1, 1945.

casionally the neck seems to bend (Fig. 4*A*, right; Fig. 3*B*, left; Fig. 6*A*, left and Fig. 7*A*) and at other time separation and displacement may develop.

If complete displacement occurs, differentiation from a purely traumatic fracture may be difficult (Fig. 8). All of our fractures were subcapital, and many presented more or less characteristic signs. One of these is a mottled zone of sclerosis of both fragments at the fracture site (Fig. 4*B*, left; Fig. 5*B*). Others failed to show these changes very clearly and the diagnosis had to be made from the history, microscopic studies or the presence of other areas of radionecrosis in the field of irradiation.

When the patient is receiving treatment through anterior fields, the pubic bones are nearer the source than are the femoral necks. They are farther away, however, when lateral and posterior fields are being used, but as Gratzek, Holmstrom and Rigler[13] have shown, they are vulnerable and often suffer considerable damage. Four of our patients show a mottled rarefying and sclerotic osteitis in both pubic bones

FIG. 6. *A*, March 19, 1943; *B*, July 16, 1943.

and in one of these, there is a fracture of the anterior ramus. In addition to fracture in the pubic ramus, this case shows dense, chalky areas in one ilium near the sacroilac joint, as well as subcapital increase in density with what we interpret as an impacted fracture in the opposite femoral neck. In one other case, we found sclerotic post-irradiation changes in the opposite hip.

TREATMENT

It is not within the scope of this paper to discuss in detail the treatment of irradiation fractures, but it seems worth while to present some of the methods used and the results obtained.

All of our patients with suspected fractures are seen by the Orthopedic Department where surgery is done when indicated. Some patients are refused operation because of their poor physical condition. Others develop a satisfactory weight tolerance and even though in their cases an operative procedure is possible, it is not done.

In 12 patients presented here, there are 16 fractures of the femoral neck. These may be classified in three general groups depending upon the degree of damage. In Group I are included those cases with incomplete fractures. There is usually a subcapital zone of sclerosis with either impaction or bending at the site of fracture. Group II consists of complete fractures with little or no displacement. Those with complete displacement,

FIG. 7. *A*, February 7, 1941, *B*; May 31, 1945.

with or without aseptic necrosis of the head, are classified in Group III. The femoral head appeared viable at the time of treatment in all of our cases. If it had not appeared so, a different plan of treatment might have been employed. In all cases of spontaneous fracture due to irradiation, it should be remembered that the blood supply is uncertain, and necrosis of the head is always a potential complication. One should keep this in mind when treating the fracture and employ some course that will provide a functioning hip regardless of the fate of the femoral head. For this reason, osteotomy is preferred in all Group III and some Group II cases.

Three of our patients had fractures that are classified in Group I. One of these was already healing when we first saw it, and since there was no disability, nothing was done. It healed satisfactorily (Fig. 4). The other 2 healed well on bed rest after which the patients were given crutches (Fig. 3*C*, right).

In Group II there are 4 fractures, one of which had internal fixation with threaded Steinmann pins (Fig. 5). It healed with firm osseous union and the patient is able to walk unaided. Three others caused little or no disability. One of them healed spontaneously (Fig. 2), while another died one year after the fracture was discovered. The third already showed early healing and since there was only slight varus deformity with no discomfort, the patient was treated with walking exercises, heat and massage. She could walk quite well with a cane after six months and roentgenograms showed some evidence of healing. There are 9 fractures in Group III. In 3 of these, subtrochanteric osteotomies have yielded good results. One patient walks unaided but the other case, a patient with bilateral fractures still uses crutches. In 2 other fractures (Fig. 3 and 7) of the same group, osteotomies of the Lorenz type were performed with excellent results. The femoral head in 1 case has become necrotic and is breaking down but the patient is able to walk unaided with little discomfort. Her main complaint is due to the shortening. Another

Fig. 8. April 12, 1944.

fracture was nailed with a Smith-Petersen nail after adequate reduction, but the nail was later extruded and absorption of the neck and non-union resulted. (Fig. 6). The other 3 fractures in this group received no treatment. One of these has not been followed while another one occurred in a patient where operation was contraindicated. Her fracture was followed for three years and did not unite (Fig. 8). The third healed spontaneously with solid osseous union (Fig. 4).

Phemister[23] states that he has observed healing in 1 case of fracture of the femoral neck following irradiation after immobilization. In another case, four months after fracture, there was a living head as proved by biopsy. He reduced the displacement, inserted three threaded wires and placed three onlay grafts of thin cortical bone taken from the upper end of the tibia across the fracture line. There was prompt healing. The head remained alive and the patient has an excellent functional result, fourteen months after operation. Barden[5] has reported a case with bilateral incomplete fractures and no displacement that was treated with bed rest and Buck's extension. Both healed with firm union and no disability.

SUMMARY AND CONCLUSIONS

1. A brief review of the literature is presented in which an attempt is made to mention the important contributions to the subject of skeletal injuries secondary to pelvic irradiation.

2. Pathologic changes in bone following intensive irradiation are considered and it is believed that the most important of these is osteoporosis. Osteoporosis secondary to vascular absorption and osteoclastic activity is probably the predisposing cause of irradiation fractures in the femoral neck.

3. Damage to the periosteum, blood vessels and bone cells is another but subordinate factor in the etiology of these lesions.

4. Twelve patients with fracture of the femoral neck following pelvic irradiation have been seen in this hospital. This is an incidence of 0.87 per cent. All patients received treatment through lateral fields and since the dose administered to the parametrium through them is about 25 per cent of the total, we think that their use should not be abandoned. We believe that the risk of serious skeletal damage has been exaggerated. The incidence of fracture is low and when it does occur the prognosis is usually good.

5. The recognition of these fractures depends upon the history and roentgenographic appearance. The latter is sufficiently characteristic to permit a presumptive diagnosis.

6. The treatment depends upon the general condition of the patient and the severity of the deformity. If no displacement has occurred, immobilization may be sufficient. If there is displacement a satisfactory result may follow reduction and immobilization, but osteotomy may be advisable in some cases.

W. P. Stampfli, M.D.
St. Luke's Hospital
Denver, Colo.

H. Dabney Kerr, M.D.
University Hospital
Iowa City, Iowa

REFERENCES

1. BACLESSE, F., and REGAUD, C. Personal communication quoted by Strauss and McGoldrick.[32]
2. BAENSCH, W. Knockenschädigung nach Röntgenbestrahlung. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, *36*, 1245.
3. BAENSCH, W. Ueber Spontanfrakturen des Schenkelhalses nach Röntgenbestrahlung. *Röntgenpraxis*, 1932, *4*, 716–718.
4. BAKER, L. D. Spontaneous fracture of femoral neck following irradiation. *J. Bone & Joint Surg.*, 1941, *23*, 354–358.
5. BARDEN, S. P. Healing of radiation fractures of necks of femora. *Radiology*, 1943, *41*, 389–394.
6. BOYD, WILLIAM. Textbook of Pathology. Fourth edition. Lea & Febiger, Philadelphia, 1943, p. 913.
7. CONZETT, D. C. Fracture of neck of femur following irradiation for carcinoma of the uterus. *J. Iowa M. Soc.*, 1943, *33*, 15.
8. DAHL, B. La théorie de l'ostéoclasie et le comportement des ostéoclastes vis à vis du bleu trypan et vis à vis de l'irradiation aux rayons X. *Acta path. et microbiol. Scandinav.*, Supp. 26, 1936, pp. 234–239.
9. DALBY, R. G., JACOX, H. W., and MILLER, N. F. Fractures of femoral neck following irradiation. *Am. J. Obst. & Gynec.*, 1936, *32*, 50–59.
10. EWING, J. Tissue reactions to radiation. AM. J. ROENTGENOL. & RAD. THERAPY, 1926, *15*, 93–115.
11. EWING, J. Radiation osteitis. *Acta radiol.*, 1926, *6*, 399–412.
12. FAILLA, G. Absorption of radium radiations by tissues. AM. J. ROENTGENOL., 1921, *8*, 215–232.
13. GRATZEK, F. R., HOLMSTROM, E. G., and RIGLER, L. G. Post-irradiation bone changes. AM. J. ROENTGENOL. & RAD. THERAPY, 1945, *53*, 62–76.
14. HEALY, W. P., and FRAZELL, E. L. Methods and results of treatment in carcinoma of cervix at Memorial Hospital. *Am. J. Obst. & Gynec.*, 1937, *34*, 593–606.
15. HIGHT, D. Spontaneous fracture of femoral neck following roentgen-ray therapy over the pelvis. *J. Bone & Joint Surg.*, 1941, *23*, 676–681.
16. JOHNSTON, ZOE A., SCHMITZ, H., COSTOLOW, W. E. Discussion of papers by Hofmann; Stevens and Payne; Howes and Strauss; Bowing and Fricke, and Pomeroy. AM. J. ROENTGENOL. & RAD. THERAPY, 1939, *41*, 76.
17. KALAYJIAN, B. S. Bilateral subcapital fractures of femurs following carcinoma of uterus. AM. J. ROENTGENOL. & RAD. THERAPY, 1938, *40*, 383–385.
18. KROPP, L. Ueber Spontänfrakturen des Schen-

kelhalses nach Röntgenbestrahlungen wegen Uteruskarzinoms. *München. med. Wchnschr.*, 1934, *81*, 214–215.

19. Miller, N. F., and Folsome, C. Carcinoma of cervix. *Am. J. Obst. & Gynec.*, 1938, *36*, 545–561.
20. Okrainetz, Clara L., and Biller, S. B. Fracture of neck of femur complicating roentgen therapy of ovarian cancer. Am. J. Roentgenol. & Rad. Therapy, 1939, *42*, 883–887.
21. Peck, W. S. Fracture of femoral neck following pelvic irradiation. *Univ. Hosp. Bull., Ann Arbor*, 1939, *5*, 33–34.
22. Peck, W. S. Personal communication.
23. Phemister, D. B. Personal communication.
24. Phemister, D. B. Fractures of neck of femur, dislocations of hip, and obscure vascular disturbances producing aseptic necrosis of head of femur. *Surg., Gynec. & Obst.*, 1934, *59*, 415–440.
25. Phemister, D. B. Changes in bones and joints resulting from interruption of circulation. I. General considerations and changes resulting from injuries. *Arch. Surg.*, 1940, *41*, 436–472.
26. Phemister, D. B. Radium necrosis of bone. Am. J. Roentgenol. & Rad. Therapy, 1926, *16*, 340–348.
27. Philipp, E. Knochenerkrankungen bei wegen Uterus karzinoms mit Röntgenstrahlen bestrahlten Frauen. *Strahlentherapie*, 1932, *44*, 363–378.
28. Quimby, E. H. Personal communication.
29. Regaud, C. Sur la sensibilité du tissu osseux normal vis-à-vis des radiations X et gamma et sur le mecanisme de l'ostéo-radio-necrose. *Compt. rend. Soc. de biol.*, 1922, *87*, 629.
30. Reggio, W., and Wilson, P. D. Fractures of region of the hip. In: Experience in the Management of Fractures and Dislocations. P. D. Wilson, Editor. J. B. Lippincott Co., Philadelphia, 1938, p. 815.
31. Slaughter, D. P. Radiation osteitis and fractures following irradiation. Am. J. Roentgenol. & Rad. Therapy, 1942, *48*, 201–212.
32. Strauss, H., and McGoldrick, J. L. Fracture neck following roentgen therapy for gynecologic malignancy. *Am. J. Obst. & Gynec.*, 1941, *41*, 915–934.
33. Truelsen, F. Injury of bones by roentgen treatment of cancer of uterine cervix. *Acta radiol.*, 1942, *23*, 581–591.
34. Zöllner, F. Die Komplikationen der Röntgenbestrahlung von Kehlkopfkarzinomen durch Tumorperichondritis und Bestrahlungsperichondritis und über die Frage der Strahlenschädigung von Knochengewebe. *Strahlentherapie*, 1941, *70*, 193–242.

DYSGERMINOMA OF THE OVARY

A REVIEW OF ELEVEN CASES OF PROVED TUMOR, WITH SPECIAL REFERENCE TO RADIOSENSITIVITY*

By ROBERT D. MORETON, M.D.
Fellow in Radiology, Mayo Foundation
and
ARTHUR U. DESJARDINS, M.D.
Section on Therapeutic Radiology, Mayo Clinic
ROCHESTER, MINNESOTA

WITH well over 300 cases of ovarian dysgerminoma reported in the medical literature, present-day clinicians and pathologists have at their command an ever-increasing volume of knowledge concerning the origin, biologic history and pathologic effects of this gynecologic "newcomer." Thanks to the pioneering work of Meyer,[12] Schiller,[17] Novak[14–16] and others,[8,10,11] it has been shown that, to quote Ewing,[6] "It [the dysgerminoma] is a malignant, radio-sensitive, cellular tumor, occurring at rather early ages, lacking hormonal effects, composed of large polyhedral cells of embryonal type, and lying in a loose stroma containing many lymphocytes." For the most part treatment has been surgical, and the success of operation has been remarkably good in cases in which the tumor was unilateral (about 60 per cent of cases) and encapsulated. Perusal of the current literature, however, reveals that, with a few exceptions, the employment of roentgen therapy has been neglected. This observation has all the more implications when it is considered that roentgen therapy has long since proved its worth in cases of seminoma of the testis, a common malignant testicular neoplasm which possesses the identical histopathologic picture seen in dysgerminoma of the ovary.

With this in mind, we wish to present data obtained from study of 11 dysgerminomas seen at the Mayo Clinic from 1905 to 1943. Eight of the cases concerned have been reported previously in detail. The present report deals primarily with the radiotherapeutic aspects. The clinical and pathologic data in the cases not reported in the text of the present paper are summarized in tabular form merely for purposes of completeness (Table 1). So far as the general historical background of the subject is concerned, the medical literature of the past ten years contains a number of reviews of the factors which led to the "discovery and isolation" of the ovarian dysgerminoma as an entity. Hence, no useful purpose would be served by reiteration. The following observations comprise some of the important features of recent studies on the problem.

HISTOGENESIS

This problem is still unsettled. Many investigators are convinced that seminoma of the testis is derived from adult spermatogonia, but that, although ovarian dysgerminoma duplicates microscopically its testicular counterpart, spermatogonia do not occur in the ovary. Proponents of the theory that ovarian dysgerminoma originates from primitive mesenchyma disregard the fact that dysgerminoma frequently is a tumor of hermaphrodites or pseudohermaphrodites. Those who trace the tumor to primitive sexual cells of an undifferentiated (or neutral) character find difficulty in explaining the occurrence of the lesion among women who are normal to the extent of being parous. In general, however, the majority of investigators believe that dysgerminoma is derived from embryonic sexual cells as a result of some undetermined stimulus. A few believe with

* Partial abridgment of thesis submitted by Dr. Moreton to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Radiology.

TABLE I

SALIENT DATA: 7 OF 11 CASES OF PROVED DYSGERMINOMA OF THE OVARY*

Case	Age, years	Before or after menopause	Pregnancies	Previous operations	Symptoms	Examination, results	Operation, roentgen treatment	Outcome
5	27	Never menstruated	0	0	"Run down" for weeks; loss of weight, strength; enlargement abdomen 9 mo.; increase size and hardness of abdomen	Dullness both lobes of lung; tense tumor filling abdomen and flanks	Subtotal abdominal hysterectomy; bilateral salpingo-oophorectomy; appendectomy. No roentgen therapy	Died 4 mo. postoperatively of recurrence
6	15	Before	0	0	Syncope associated with periods for 2 years; constipation; tumor noticed at 8 mo. of age; irregular menses for 2 mo.	Hard nodular tumor from pelvis to navel; tumor movable and nontender	Total abdominal hysterectomy; bilateral salpingo-oophorectomy. No roentgen therapy	Died 1 day postoperatively of cardiorenal failure
7†	24	Before	2	0	Numerous "female complaints"; pain in pelvis; blood-tinged discharge; palpitation	Large mass in lower half of abdomen; more on left. Abdomen tender	Subtotal abdominal hysterectomy; bilateral salpingo-oophorectomy. No roentgen therapy	Well 13½ yr. postoperatively on Oct. 15, 1941
8†	18	Before	0	Thyroidectomy 4 years before	Mass in side for 2 yr.; enlarging lately; pain in back and abdomen	Tumor of right ovary found at time of thyroidectomy; pelvic mass then extended to right and to umbilicus	Total abdominal hysterectomy; bilateral salpingo-oophorectomy; appendectomy. No roentgen therapy	Died 6 days postoperatively in presence of peritonitis and bronchopneumonia
9†	19	Before	0	0	Pelvic pain; irregular menses	Uterus hard, small and irregular; pedunculated tumor halfway to navel, more on the left	Subtotal abdominal hysterectomy; bilateral salpingo-oophorectomy; appendectomy. No roentgen therapy	Well 14 yr. postoperatively on Sept. 12, 1941
10†	18	Before	0	Appendectomy	Pain in region of left kidney; small lump in lower left abdominal quadrant; weight loss	Smooth, firm, movable mass from pelvis to navel; more to right	Right salpingo-oophorectomy. No roentgen therapy	Periods normal; well 11 yr. postoperatively; 2 operations since; no evidence of metastasis
11†	29	Before	0	0	Onset 2 mo. previously with cold; low-grade fever; mass found and pregnancy considered; later, pelvic mass thought more than pregnancy; vomited daily	Rounded movable, slightly tender abdominal mass; uterus slightly enlarged	Right salpingo-oophorectomy; no roentgen therapy	Well 5½ yr. postoperatively; 1 child born; 1 miscarriage; periods normal

* The 4 cases required to bring the total to 11 are reported in detail in the text of this paper.
† Previously reported by Dockerty and MacCarty, 1939.[3]

Ewing that the tumor is an atypical teratoma; other workers accept Fischel's[8] theory in which the origin of the lesion is said to be related to totipotential mesenchymal cells.

MALIGNANCY

Few investigators still believe, with Schiller, that many of these tumors are benign. Regardless of the size or extent of the lesion, the microscopic structure is so characteristic as to allow easy identification in practically all cases. As is true of seminoma of the testis, the picture in dysgerminoma is that of anastomosing strands and islands of large round cells with hyperchromatic nuclei and large nucleoli. Interspersed with these cells are clusters of lymphocytes and occasional giant cells. As field after field

is examined the picture becomes monotonously similar, excepting as areas of necrosis and hemorrhage are encountered. In the ovary, as in the testis, the histopathologic diagnosis is that of a grade 4 (Broders' classification) embryonal carcinoma with lymphoid stroma. Paradoxically, however, this particular type of grade 4 lesion is associated with a relatively good clinical prognosis in those instances wherein the neoplasm is unilateral, encapsulated and nonadherent.

RESULTS OF HORMONAL STUDIES

The early observation of Robert Meyer that dysgerminoma occurred most frequently among young pseudohermaphrodites and that dysgerminoma is the lesion which most frequently attacks the gonads of such an individual gave rise to early suspicions that the tumor influenced the host by means of some hormone. It next became apparent that Meyer's series of cases was not representative, and that many patients who had the neoplasm in question were not pseudohermaphrodites. Moreover, it was discovered that removal of a dysgerminoma in no way affected the pre-existing condition of pseudohermaphroditism. Bell,[1] Geist,[9] Klaften,[10] Novak and Gray[15] concluded that hormonal secretion cannot be attributable to this tumor. Results of recent studies, however, have shown that dysgerminoma may be associated with an excess of anterior pituitary-like substances in the urine.

In one of our own cases, the urine of the patient gave a positive reaction to the Friedman test; this reaction became and remained negative after surgical removal of the tumor. Such an observation parallels the experience of Ferris,[7] who demonstrated that seminoma of the testis is not by any means without hormonal effect. The observation does not circumvent the argument of Ewing that such a result indicates a teratomatous origin of the tumor and association with chorionepithelioma.

RADIOSENSITIVITY

Most writers have said that dysgerminoma reacts to roentgen rays in a characteristic way. The tumor has been classed as being radiosensitive by such authors as Stewart and Farrow,[19] Dudman,[5] Dockerty and Masson,[4] and others. Novak and Gray, however, stated that the question of whether or not dysgerminoma really can be regarded as radiosensitive cannot be answered definitely, and they added that some authors—one being Meyer—think it is radioresistant, whereas others think it is radiosensitive. The most striking results have been presented by Seegar,[18] who reported that 3 patients received substantial benefit from roentgen therapy. One of the patients received repeated doses of high voltage roentgen rays over a period of five years for a recurrence of the neoplastic process in the retroperitoneal lymph nodes, and had been well for twelve years at the time of Seegar's report. The other patients had survived five and three years, respectively, after irradiation.

REPORT OF CASES

Eight of the 11 cases concerned in this study were reported in 1939 by Dockerty and MacCarty,[3] who were not primarily occupied by the roentgen therapeutic aspects of the problem. We shall report herein, as Cases I, II and III, 3 of the cases of Dockerty and MacCarty. The fourth case to be reported herein has not previously been published by anyone.

CASE I (Case 4 of Dockerty and MacCarty). A white married woman, aged twenty-nine, registered at the Mayo Clinic on April 4, 1917, complaining of a lump in the abdomen. She said that menstruation had begun when she was fifteen years old, and it had been regular until six months prior to her registration. She had borne two children and had had one miscarriage. The rest of this patient's history and of her familial history was irrelevant.

The patient had undergone myomectomy and appendectomy, performed elsewhere. Eight months after these operations she had begun to experience pains "all over" the back and down her thighs. For four months menstruation had been irregular. She had noticed small pieces of fleshlike material in the vaginal discharge. A month before registration, this patient had discovered a lump in the abdomen.

Pelvic examination disclosed a nodular type of uterus and a mass situated in the region of the adnexa uteri on the left. On April 10, 1917, a Wertheim type of hysterectomy was performed and the adnexa on both sides were removed because of a solid tumor of the left ovary. Several iliac lymph nodes were found to be indurated; they were removed. The pathologist reported a solid neoplasm of the left ovary measuring 4 by 3 by 2 cm. The mass was adherent to the uterus, the serosal surface of which contained several small metastatic nodules. Small iliac lymph nodes 1 to 2 cm. in diameter were manifestly malignant. The soft, brainlike consistency of these nodes was, like that of the primary ovarian neoplasm, typical of the pathologic picture of dysgerminoma. Postoperative roentgen therapy was administered through four anterior abdominal fields and two posterior abdominal fields. The factors were as follows: 135 kv., 5 ma., 3 mm. aluminum filter; distance 9 inches; time, six to six and a half minutes. The patient was advised to continue to receive roentgen therapy in her home city. She enjoyed good health after she left the Clinic, and was still well when last heard from, seventeen years later.

Case II (Case 7 of Dockerty and MacCarty). A white girl, aged twelve, registered at the Mayo Clinic on October 28, 1927, complaining of enlargement of the abdomen that had been present for two years. Seven days prior to registration she had begun to suffer from pain in the lower part of the back. A physician in her city had palpated a mass in the lower part of the abdomen and another mass in the upper and middle portion of the abdomen.

Results of physical examination at the Clinic confirmed the observations of this patient's home physician. On November 5, 1927, total abdominal hysterectomy was performed, with removal of the adnexa uteri on each side. Each ovary was found to be the size of a grapefruit (about 17 to 10 cm. in the case of one and 12 by 9 cm. in the case of the other). The right ovary was free, and lay in the upper part of the pelvic cavity; the left ovary was adherent in the pelvic cavity. Because of the many adhesions, complete removal of the neoplastic processes was effected only with the greatest difficulty. The report of the pathologist was sarcoma of the ovaries. The histopathologic picture of both neoplasms of the ovaries was typical of dysgerminoma.

This patient received postoperative roentgen therapy administered to the pelvis through four anterior fields and two posterior fields. Each field received a total dose of approximately 520 r. The patient was advised to arrange for a second course of roentgen therapy within a month's time, but she did not do so. She died at home three months later, after the occurrence of symptoms of intestinal obstruction possibly caused by a recurrent malignant process.

Case III (Case 9 of Dockerty and MacCarty). A white girl, aged fifteen, registered at the Mayo Clinic on July 5, 1935, complaining of painful swelling of the abdomen of ten weeks' duration. She had been admitted to a hospital, where 2 gallons (7.6 liters) of pink fluid had been removed from the abdomen. Ascites had recurred; paracentesis had been repeated five weeks later and then once later. The fluid obtained at the last two tappings had been dark red. The patient said that menstruation always had been irregular, associated with dysmenorrhea, and that the flow had been scanty. Her last menstrual period had ended two months before she registered at the Clinic.

On examination, a tumor was palpated in the pelvic cavity. Ascites of extreme degree was present. On July 12, 1935, total abdominal hysterectomy and bilateral salpingo-oophorectomy were performed because of dysgerminoma of the right ovary. Many adhesions were noted. The ascitic fluid was bloody. The pathologist reported that the uterus, tubes and right ovary were infantile.

After operation, the patient received roentgen therapy administered through four pelvic fields. The factors were as follows: 135 kv., 5 ma., 6 mm. aluminum filters; distance 16 inches; time, twenty-eight minutes, for a total dose of 500 r to each field. The patient was advised to receive additional roentgen therapy in her home city, but she did not do so.

One month after she had been dismissed from the Clinic, the patient reported that she was in "fine" condition. It appears that a month later (September), however, "bloating in the region of the stomach" occurred and that the patient became short of breath. She died on October 16, 1935, three months after the surgical treatment, probably of recurrence of the malignant process.

Case IV. A married white woman, aged twenty-three, registered at the Mayo Clinic on June 10, 1940, complaining of enlargement of the abdomen which she said had been present

for seven months. After the normal delivery of a child on November 10, 1939, the abdomen had remained abnormally large. Menstruation had returned a month after delivery, and except for some irregularity in the two periods prior to her registration, the periods had been normal. Two months before registration, the patient had noticed progressive enlargement of the abdomen, and two weeks before registration she had noticed a lump in the left side of the neck.

Examination disclosed three large, fixed lymph nodes in the left supraclavicular region. In the abdomen a tumor the approximate size of a seven months' pregnancy was palpated. It seemed to lie more on the left side than on the right. A Friedman hormone test for pregnancy gave a positive result.

On June 17, 1940, a lymph node from the left supraclavicular region of the neck was removed. Later, total abdominal hysterectomy and bilateral salpingo-oophorectomy were performed. The left ovary contained a large solid carcinoma which, grossly, appeared to be a dysgerminoma.

The pathologist reported that the excised lymphatic vessels contained a grade 4 adenocarcinoma which was metastatic and similar to the neoplasm of the ovary. The left ovary contained a hemorrhagic, necrotic, grade 4 adenocarcinoma (dysgerminoma). The right ovary was affected by cystic oophoritis. The uterus was myomatous; the endometrium, atrophic.

On July 6, 1940, the patient began to receive a course of postoperative roentgen treatment to the pelvis through four anterior fields and four posterior fields, and to the left supraclavicular area through one field. The factors were: 130 kv. (constant potential), 6 mm. aluminum filtration; distance, 16 inches; time, twenty-one minutes, or a total dose of 540 r to each field.

The patient returned two months later, on September 9, 1940. Her condition had improved greatly. Results of abdominal examination were essentially negative, and the previously enlarged lymph nodes in the neck could scarcely be palpated. The patient then received a second course of roentgen treatment, arranged much like the first course. She returned again on March 13, 1941. By that time, she had gained 16 pounds (7.3 kg.) and felt well. The patient was examined again in 1945, five years after operation. She reported that she had nothing of which to complain, and she was found to be normal.

ROENTGENOLOGIC CONSIDERATIONS

At present, according to general opinion, only one kind of treatment is indicated for probable or actual metastasis which follows surgical removal of the primary tumor. This treatment is radiotherapy, best carried out with roentgen rays. The use of radium should be limited to small zones of metastasis.

Since the advent of protracted, fractional irradiation with roentgen rays, this method or some modification of it has achieved favor among most therapeutic roentgenologists. In a large number of institutions, as pointed out by Nash and Leddy,[13] this method has entirely replaced the single-dose method in the treatment of malignant neoplasms. The efficacy of protracted, fractional irradiation of epidermoid carcinomas of certain regions of the body, particularly of the pharynx or larynx, is generally admitted. However, reasonable arguments against the use of this method in the treatment of dysgerminoma and its metastatic processes can be advanced. Those who are familiar with the reaction of the mucous membranes during the roentgenologic treatment of carcinoma of the pharynx can appreciate the reaction which must take place in the peritoneum and gastrointestinal tract; perhaps the reaction that is produced in the sensitive mucosa of this tract is even more severe than that produced in the mucous membranes of the pharynx.

In the classification advised by one of us (Desjardins)[2] of the order of radiosensitiveness of various body cells and tissues, the epithelial cells of the testis and ovary have been given a high rating. Also, as we have already pointed out herein, Stewart and Farrow and others have stated that the so-called seminoma of the ovary has a high degree of radiosensitiveness. Nash and Leddy compared the great radiosensitiveness of seminoma of the testis, which is the same as that of corresponding tumors of the ovary, with that of lymphoblastoma. So far as we have been able to determine on the basis of the contemporary radiologic literature, it seems certain that in many

large centers roentgen treatment for lymphoblastoma is not carried out according to the protracted fractional period. By analogy, this may be an argument against the use of this method of treatment for dysgerminoma. Nash and Leddy thought that this reasoning was valid in relation to the treatment of similar tumors in the male.

Since both ovarian dysgerminoma and seminoma rank next to lymphoblastoma in radiosensitiveness, and since treatment with roentgen rays generated at higher potentials may result in intensive, unsafe, and perhaps unncessary irradiation of unaffected abdominal structures, the treatment of dysgerminoma in our series has been carried out largely with roentgen rays generated at 130 kv. (constant potential) usually filtered through 6 mm. of aluminum, having a half-value layer equivalent to 0.32 mm. of copper.

The method now used at the Mayo Clinic is to treat through four anterior (abdominal) fields which extend from the xiphoid cartilage to the pubic bone, and through four corresponding posterior fields. The left supraclavicular space is also routinely irradiated. The surface of each field receives a dose of 540 r in air. The amount of treatment administered each day depends on the patient's tolerance, and especially on the state of the formed elements of the blood. The treatment is repeated in a month, even though the patient may be clinically well. Subsequent treatment is governed by the results of periodic examinations.

The available evidence does not indicate that dysgerminoma reacts more favorably to treatment with rays generated at 200 kv. than to treatment with rays generated at 130 kv. (constant potential).

There are two chief reasons for the arrangement of treatment that has been outlined herein. Both are based on the peculiarities of the neoplasm in question. First, since the growth of this tumor is characteristically rapid, the tumor may spread anywhere in the abdominal cavity, and this extension of the malignant process may not be detected until later. This is illustrated by our Case III, reported herein, in which treatment was administered through four pelvic fields. Three months later the patient died of obstruction high in the abdominal cavity, which apparently had not been included in the fields of treatment.

The second reason for the arrangement of treatment as outlined herein is that these particular tumors are known to spread through the lymphatic vessels. Sometimes they may spread far and wide, as in our Case IV, reported herein, although in this case metastasis through the blood stream, by way of the lungs, cannot be ruled out. Metastasis also was widespread in a case reported by Klaften,[10] in which lymph nodes in the neck were removed. Other examples of metastasis through the lymphatic vessels may be found in our Case I and in Case 6 in Table I. Case 6 in Table I serves to bring out the importance of postoperative irradiation such as has been outlined herein, because, although clinical or surgical signs of extension of the malignant process were not found, necropsy carried out one day after operation revealed involved para-aortic lymph nodes.

Seegar drew attention to the fact that instances of metastasis to the lungs from ovarian dysgerminoma had not been reported in the literature. This observation was corroborated by the observations made in our series of cases. For this reason, the field over the mediastinum, use of which was advocated by Nash and Leddy in the treatment of seminoma of the testis, has not been employed in the irradiation of ovarian dysgerminoma. Pathologically, however, there is no reason why mediastinal and pulmonary involvement could not take place in patients who have ovarian dysgerminoma, especially when widespread metastasis occurs, such as did occur in the case we have reported. Also, it is well known that seminoma metastasizes to the mediastinum and to the lungs. Hence, should metastasis of an ovarian dysgerminoma to the lungs be found or reported, mediastinal and pulmonary irradiation should be included, just as it is in the treatment of seminoma.

SUMMARY AND CONCLUSIONS

Eleven cases of proved ovarian dysgerminoma have been reviewed from the standpoint of roentgen therapy. Four cases are reported in detail. Of the whole series of eleven cases, eight were reported in 1939 by Dockerty and MacCarty, but without regard for the roentgen therapeutic aspects of the disease in question.

The problem of the histogenesis of ovarian dysgerminoma is still unsettled, but the fact that the lesion develops in otherwise normal women or that it can even be associated with pregnancy is illustrated by the present series. Histopathologically, the structure of ovarian dysgerminoma is that of a grade 4 (Broders' classification) embryonal carcinoma with lymphoid stroma. The clinical outcome apparently is determined by such factors as the presence of bilateral lesions, whether or not the capsule of the neoplasm is intact, whether or not the tumor has twisted on its pedicle and other considerations. Results of recent studies have shown that ovarian dysgerminoma can be associated with the presence of an excess of anterior pituitary-like substance in the urine. Ovarian dysgerminoma has a high degree of radiosensitiveness, being comparable in this respect to seminoma of the testis, which is its counterpart among male patients. Except in special instances in which childbirth has to be considered, the treatment which offers the best prospect of longevity is surgical removal of the tumor plus adequate postoperative therapy. At present, the only treatment for probable or actual metastasis is radiotherapy, which is best carried out with roentgen rays. The moderate voltage, single-dose method, as has been outlined herein, is preferred.

Arthur U. Desjardins, M.D.
Mayo Clinic
Rochester, Minnesota

REFERENCES

1. BELL, E. T. A Textbook of pathology. Fourth edition. Lea & Febiger, Philadelphia, 1941, pp. 392–394.
2. DESJARDINS, A. U., Radiosensitiveness of cells and tissues, and some medical implications. *Arch. Surg.*, 1932, *25*, 926–942.
3. DOCKERTY, M. B., and MACCARTY, W. C. Dysgerminoma, with report of nine cases, one associated with pregnancy. *Am. J. Obst. & Gynec.*, 1939, *37*, 878–885.
4. DOCKERTY, M. B., and MASSON, J. C. Clinical features, types and grades of malignant ovarian tumors. *S. Clin. North America*, 1941, pp. 1201–1210.
5. DUDMAN, V. E. Dysgerminoma and arrhenoblastoma. *West. J. Surg.*, 1939, *47*, 128–135.
6. EWING, JAMES. Neoplastic Diseases, a Treatise on Tumors. Fourth edition. W. B. Saunders Co., Philadelphia, 1940, pp. 661–662.
7. FERRIS, D. O. A study of the pathology of tumor of the testicle in correlation with the hormone findings in the excreted urine. Thesis, Graduate School, University of Minnesota, 1941, 43 pp.
8. FISCHEL, ALFRED. Über die Entwicklung der Keimdrüsen des Menschen. *Ztschr. f. Anat. u. Entwcklngsgesch.*, 1930, *92*, 34–72.
9. GEIST, S. H. The histogenesis of certain ovarian tumors and their biologic effects. *Am. J. Obst. & Gynec.*, 1935, *30*, 650–664.
10. KLAFTEN, E. Beitrag zur Klinik des Disgerminoma ovarii. *Zentralbl. f. Gynäk.*, 1933, *57*, 736–741.
11. KLAFTEN, E. Weiterer Beitrag zur Klinik des Disgerminoma ovarii (Robert Meyer). *Arch. f. Gynäk.*, 1934, *158*, 544–549.
12. MEYER, ROBERT. The pathology of some special ovarian tumors and their relation to sex characteristics. *Am. J. Obst. & Gynec.*, 1931, *22*, 697–713.
13. NASH, L. A., and LEDDY, E. T. Seminoma of the testis from the standpoint of roentgen treatment. AM. J. ROENTGENOL. & RAD. THERAPY, 1943, *50*, 162–196.
14. NOVAK, EMIL. Ovarian tumors of endocrine nature. *J.A.M.A.*, 1941, *116*, 947–950.
15. NOVAK, EMIL, and GRAY, L. A. Dysgerminoma of the ovary; clinical and pathologic study of 17 cases. *Am. J. Obst. & Gynec.*, 1938, *35*, 925–937.
16. NOVAK, EMIL, and LONG, J. H. Ovarian tumors associated with secondary sex changes; granulosa cell carcinoma and arrhenoblastoma. *J.A.M.A.*, 1933, *101*, 1057–1063.
17. SCHILLER, WALTER. Recent findings in solid ovarian tumours. *J. Obst. & Gynaec. Brit. Emp.*, 1936, *43*, 1135–1144.
18. SEEGAR, G. E. Ovarian dysgerminoma. *Arch. Surg.*, 1938, *37*, 697–725.
19. STEWART, F. W., and FARROW, J. H. The radiosensitivity of tumors. In Pack, G. T., and Livingston, E. M.: Treatment of Cancer and Allied Diseases, by One Hundred and Forty-seven International Authors. Paul B. Hoeber, Inc., New York, 1940, vol. 1, chap. 8, pp. 98–143.

Vol. 57, No. 1

QUANTITATIVE DETERMINATION OF THE RADIUM CONTENT OF THE HUMAN BODY AND OF THE RADON CONTENT OF BREATH SAMPLES FOR THE PREVENTION AND CONTROL OF RADIUM POISONING IN PERSONS EMPLOYED IN THE RADIUM INDUSTRY

By VICTOR F. HESS *and* WILLIAM T. McNIFF
Fordham University
NEW YORK, NEW YORK

WITHIN the past few years, real progress has been made in the development of very sensitive methods for the detection of minute quantities of radium and other radioactive substances in the bodies of living persons. Robley D. Evans[1] and his associates have made outstanding contributions in this field and his methods are now widely used for routine measurements all over this country.

In the case of radium, he established the fact that, on the average, about 55 per cent of the radium in the body can be detected by its gamma-ray effect outside of the body, while 45 per cent is detected as radon in exhaled breath. In order to get an exact knowledge of the total amount of radium, therefore, two determinations have to be made:

(1) Test of the exhaled air for radon and computation of the corresponding amount of radium. According to Evans, a quantity of 1×10^{-12} curies of radon per liter of expired air corresponds to about 0.09 microgram of radium in the body (mainly in the lungs and the blood stream).

(2) Determination of the additional quantity of radium affixed to the bones and the tissues and, therefore, not contributing to the exhalation of radon.

Radon tests of exhaled air samples are relatively simple and the methods used at present are sufficiently sensitive for detection of as little as 0.1 micromicrocurie per liter, which is considered as one-tenth of the tolerance limit agreed upon by a committee organized by the National Bureau of Standards in Washington, D. C., in 1941.

For the determination of the radium affixed to the body (with no radon given off), the situation is far less satisfactory.

Gamma-ray counting tubes carefully calibrated and arranged at a distance of 1 meter from the body of the patient were used by Evans for this purpose and he summarizes his experience as follows:

> When used for routine measurements of humans, the ultimate sensitivity of gamma ray observations is about 0.2 micrograms of radium as radium C. . . . This corresponds to about 0.4 micrograms of total radium burden within the body. This value is four times the present accepted tolerance. Therefore, routine gamma ray measurements are of no practical assistance in detecting sub-tolerance quantities of radium. However, in patients whose radium burden is larger, gamma ray measurements, combined with breath radon analyses, are essential in making accurate measurements of the total radium burden and in monitoring the effects of therapy.

In view of this situation, it seemed important to us to try pushing the sensitivity of gamma-ray measurements to their limit and also to make the radon tests of breath samples easier for routine checks of workers in the radium and luminous paint industry.

I. IMPROVEMENT OF THE GAMMA-RAY METHOD

Gish and Sherman, and Hess[2] devised a compensating ionization meter which was used in the past two years for determination of the ionization by cosmic rays and by gamma rays from the soil, from rocks

and from radioactive substances floating in the atmosphere.

When the instrument was used indoors, it was soon recognized that the remarkable constancy of the background ionization would be very valuable when using the instrument for gamma-ray determination of very minute quantities of radium. A set of

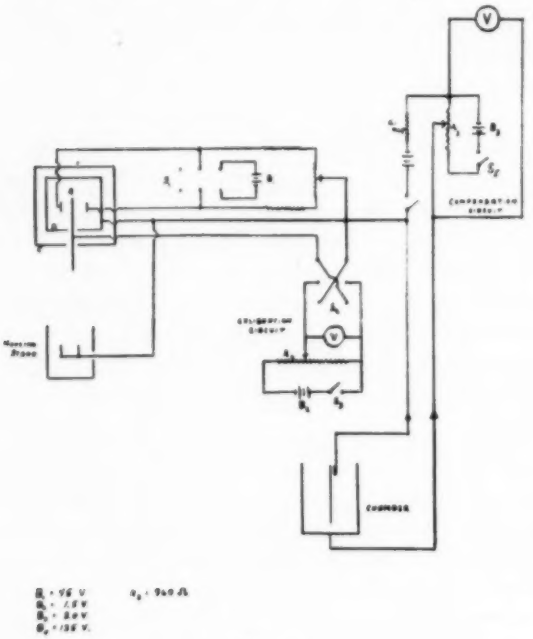


FIG. 1

secondary radium standards was developed for this purpose, ranging from 1 to 100 micrograms of radium.

The instrument itself consists of a set of three airtight ionization chambers, filled with dry nitrogen at atmospheric pressure. The chambers are evacuated and then filled with dry aged nitrogen through a cotton filter, thus eliminating all nuclei and ordinary dust particles. The chambers are cylindrical; the walls are made of brass, silver-soldered. All three chambers are geometrically similar, having a height exactly four times the radius. The volume of the largest chamber is 13 liters and only this was used for the purpose discussed here.

Before soldering the chambers, all parts were polished with fine emery paper, to remove any possible surface contamination. Then the chambers were rinsed first with dilute sulfuric acid, then with a weak cyanide solution and flushed with hot water.

Leakage of the chamber can easily be detected by the surface condition of the piece of sodium metal which is kept in a small tube connected with the chamber. If moisture seeps in, the sodium is converted into Na (OH).

The apparatus was constructed in the Department of Terrestrial Magnetism (Carnegie Institution, Washington, D. C.). It combines features of the instruments used in the early days of research on the penetrating radiation, by Wilson[3] and Compton:[4] the guard ring of the ionization chamber is kept at practically the same potential (135 volts) as the inner electrode (connected with the needle of the Lindemann electrometer), thus avoiding any leakage across the insulator even under adverse atmosphere conditions. The other feature is the compensation circuit: the ionization chamber can be evacuated and filled through a Hoke needle valve from a high pressure tank. The height of the cylinder is 40.6 cm., the diameter 20.3 cm.; the wall consists of brass tubing 2.5 mm. thick. The inner electrode is a cylindrical rod, with its end at a distance of 10.1 cm. from the bottom. It is permanently connected with the needle of a Lindemann electrometer which serves here only as a null instrument. The housing of the electrometer is also permanently connected to a battery of 135 volts and the quadrants are at plus or minus 22.5 volts (C battery) above or below 135 volts (see Fig. 1, wiring circuit).

The electrometer is enclosed in an outer housing which is carefully kept dry by an attached drying tube containing a piece of sodium. The outer housing, the wall of the ionization chamber and one terminal of the 135 volt battery are grounded.

If the electrometer is set up on top of the chamber, a fine steel needle contact establishes the connection between the electrometer needle and inner electrode. At the

same time, a hand-operated needle contactor connects the needle and inner electrode with the 135 volt battery. As soon as this contactor is withdrawn (at the time T=0), the needle begins to move and the rate of drift of the needle is proportional to the saturation current flowing between the grounded wall and inner electrode of the ionization chamber.

Instead of observing, as usual, the drift of the needle per unit time ("drift method"), it is much more accurate to add gradually a compensating potential of from 0 to −3 volts to the guard ring potential ("sweeping potential") by connecting the guard ring (and the inner housing of the electrometer) with a potentiometer circuit and adjusting this added voltage by turning the knob of the potentiometer continuously so as to keep the electrometer needle at its zero position. This compensating voltage is read on a Weston precision voltmeter in successive intervals of a few minutes.

This compensating voltage, divided by the number of seconds elapsed (since the withdrawal of the contactor) and multiplied by a carefully determined calibration factor, gives directly the ionization (q) in terms of the number of ions (I) produced in the chamber per cm.^{3} and per sec.

Indoors the average ionization observed with the 13.1 liter chamber amounted to 7 to 9 I (ion pairs per cm.^{3} at atmospheric pressure). It is produced (*a*) by the gamma rays from radioactive substances in the walls of the room (about 5 I), (*b*) by cosmic rays (about 2 I), (*c*) by the residual small alpha-ray ionization from the inner surface of the brass walls of the chamber (about 1 I).

If the chamber is set up outdoors, the ionization drops to about 6 I (since there only the gamma rays from the radioactive substances in the ground and air and cosmic rays are ionizing). By placing the whole apparatus over water (on a wooden pier in New York), we determined the ionization by cosmic rays plus residual ionization as 3.2 I (the very small ionization contributed by the radioactive decay-products in the atmosphere is included in this figure).

Indoors the total ionization observed changes very little; in the course of a day, the changes observed for each fifteen minute period of observation are usually less than ±0.1 I.

Therefore, it seemed feasible to use the compensation method for determining the radium content of the human body where the gamma-ray ionization of less than the accepted tolerance limit of 0.1 microgram of radium should be detectable.

Since most of the radium is lodged in the thorax of persons with incipient radium poisoning, it was thought best always to have the patient sitting upright at a standard distance (35 cm.) from the vertical axis of the ionization vessel, first with his back and then with his chest toward the chamber.

Preliminary experiments with persons employed in the radium industry showed that a number of precautions are necessary in order to get reliable results. First of all, the clothing and the shoes of such workers show often enough a contamination large enough to falsify the results. Then the skin (especially the hands) and the hair sometimes retains traces of radium dust and this again would mislead the observer.

While the presence of traces of radium on the skin and in the hair is certainly not harmless, it should not be counted in a determination of the amount of radium incorporated in the body of the patient. Lethal effects of radium poisoning are due to the fixation of inhaled or ingested quantities of radioactive substances to certain organs (lungs, etc.) and the bones of the human body. Therefore, when a person is to be tested for radium in the body, it was found necessary: (1) to have this person take a bath and have the hair shampooed several hours before the test; (2) to forbid this person to enter a radium laboratory or factory in the twelve hour period preceding the test; (3) to make this person change clothes and remove shoes (newly laundered hospital clothes should be kept ready for the purpose).

With these precautions taken, the amount of gamma radiation detected can be ascribed to the actual presence of radium (or other radioactive substances) in the body of the patient (Evans[1]).

It is clear that in gamma-ray tests, as described here, one assumes tacitly uniform distribution of the radium all over the head, thorax and abdomen, while radium lodged in the arms or legs is producing a lesser effect on account of the greater distance from the center of the chamber.

This, however, is, in our opinion, not a serious disadvantage, since the deadly cumulative action of the rays is certainly taking place in the main organs of the body and not in the extremities.

Schlundt and others[5] used essentially the same technique as we did, but their gamma-ray electroscope was antiquated and much less sensitive. The use of gamma-ray counters, as recommended by Evans,[1] is rather cumbersome, if one intends to determine the entire radium content of a living person, since it necessitates counting in many different positions.

The gamma-ray ionization chamber method used by us is simpler in operation (because it integrates the effect) and more sensitive. This will be proved below.

Experiments with a Water Phantom and the Determination of the Minimum Amount of Radium Detectable with the Gamma-Ray Method. If a known point-shaped radium preparation is used as a source of gamma rays, the ionization produced by gamma rays (RaC'') is given by the formula

$$q = K\frac{m}{r^2}e^{-\mu d}$$

where m is the amount of radium (in grams) in the sealed tube, r the distance (in cm.) from the center of the ionization chamber, μ the absorption coefficient in the medium between source and chamber and d its thickness. If only air is between chamber and the source, the term $e^{-\mu d}$ is negligible for distances of a meter or so ($\mu = 4.5 \times 10^{-5}$ cm.$^{-1}$).

The so-called "Eve's constant" K was determined for our apparatus by Hess and Balling[6] (1945) and found to be 4.8×10^9 I (pairs of ions per g Ra. per cm.^{3} sec.) at unit distance. Therefore, a preparation of 0.1 microgram of radium (10^{-7}g) at a distance of 30 cm. in air would produce an ionization of

$$q = 4.8 \times 10^9 \frac{10^{-7}}{900} = 0.5 \text{ I}$$

which is easily detectable with our instrument.

Assuming an even distribution of radium all over the thorax, one can get a calibration of the apparatus in terms of the total amount of radium distributed in the body by measuring the ionization produced by a small sealed radium tube, if this tube is immersed in a water tank of the shape of the human thorax (water phantom) at different depths and distances. We took the average of the experimental results at all distances and depths. This calibration carried out with the "water phantom" will be accurate enough since the density of the human body is almost the same as the density of water.

Such experiments were performed several times by using a standardized radium tube containing 4.15 micrograms of radium. It was found most convenient to choose a distance of 35 cm. between the center of the water tank and the center of the ionization chamber. The height of the water tank was 40 cm.; its cross section was an ellipse of 30×20 cm. Wooden cross bars with equidistant holes provided nineteen different fixed positions of the radium tube, which could easily be reproduced in subsequent experiments. A glass tube 1 mm. thick closed on one side could be placed in any of the nineteen holes in the cross bars and this allowed us to place the radium preparation at various depths below the water level of the tank. Three levels (near the top, center and the bottom of the tank) were chosen so that a complete calibration represented the average of the gamma-ray ionization produced at fifty-seven different positions of the radium tube within the body of water.

In Figure 2, the numbers indicate the position of the holes in the wooden cross bars. Table I gives the mean values of ionization (after subtracting the background) produced by the 4.15 microgram radium tube placed in these positions, at three levels within the water of the tank.

The amount of absorption of the gamma rays in the water is clearly seen from Table II where the ionization produced by the radium tube (4.15 micrograms) is listed individually for three positions (nearest, middle and farthest) in the water tank at the three levels (see Fig. 2), after subtracting the background.

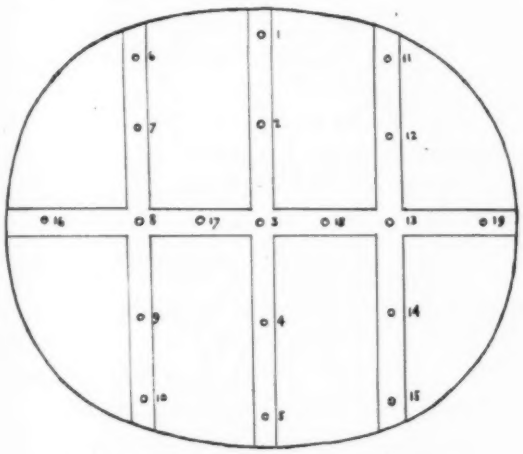


Fig. 2. Water phantom experiment.

The final figure (3.34 I per microgram) is somewhat higher since the positions No. 1, 2, 3 (Fig. 2) are all in the medium vertical plane, but, of course, the integral effect is more accurate by the calibration in fifty-seven different positions, as represented in Table I. The order of magnitude is the same.

If the background ionization taken before and after the actual measurement of the gamma-ray effect of the patient differs only by 0.1 I, it is obvious that a quantity of 1/29 microgram of radium in the body can be detected and this is much less than the accepted tolerance limit of 0.1 microgram.

The experiments carried out with a number of technicians and chemists employed in the radium industry showed that the method is simple and easy for routine work. The entire procedure (two background measurements before and after the test and two measurements with the patient sitting in front of the chamber first with the back, then with the chest toward the apparatus) takes only one hour.

The method has been in practical use for over one year and no difficulties have been encountered. The great advantage of the method is the constancy of the sensitivity and of the background. It must be emphasized that all these tests were made in a building at Fordham University where no open radium preparations are ever used.

Table I

Experiments with 4.15 micrograms of radium in the "water phantom" tank

(Distance 35 cm. between tank and center of ionization chamber. Background ionization (9.2 I) already subtracted)

	Position	Ionization	Average
Upper Plane	1, 2, 3, 4, 5	13.6 I	12.5 I
	6, 7, 8, 9, 10	12.8 I	
	11, 12, 13, 14, 15	12.8 I	
	16, 17, 18, 19	10.8 I	
Medium Plane	1, 2, 3, 4, 5	13.3	12.6
	6, 7, 8, 9, 10	12.9	
	11, 12, 13, 14, 15	13.0	
	16 to 19	11.4	
Lower Plane	1, 2, 3, 4, 5	11.7	11.05
	6 to 10	11.0	
	11 to 15	11.0	
	16 to 19	10.5	
Total mean (for 4.15 micrograms radium)			12.03 I
per microgram radium			2.90 I

Table II

Position	Upper Level	Medium Level	Lower Level	Average
No. 1	5.9 I	5.9 I	5.7 I	5.83
2	11.6 I	11.6 I	11.5 I	11.57
3	24.9 I	24.4 I	21.9 I	23.77
General average				13.7 I
Per microgram of radium				3.34 I

Correction for Wall Effect. It is evident that a person sitting very close to the ionization chamber will absorb part of the local gamma rays coming from the walls of the room; this holds for the observer as well as for the person who is examined.

Evans[1] therefore recommends having a non-contaminated person sitting in exactly the same position relative to the measuring instrument for an exact determination of the background ionization.

We preferred to determine the screening effect of the patient on the local radiation from the walls of the room directly; for this purpose, we made a series of determinations of the ionization in the chamber (1) without patient, (2) with a patient sitting close to the chamber (at a distance of 35 cm. from the center of the body of the patient to the center of the chamber).

Between March, 1945, and April, 1946, three extensive series of such tests were made with persons from 150 to 200 pounds in weight. Each series consisted of at least ten determinations both with and without a person in front of the chamber. The mean differences were 0.370 I, 0.364 I and 0.380 I. The average 0.37 I was chosen as the final average correction in a laboratory room at Fordham University used for a person of medium weight and thickness. The application of this correction may be illustrated by an example:

Background observed (ground floor)	7.27 I ±0.1 I
Background corrected for the case of an inactive patient sitting in front of the apparatus (0.37 I)	6.90 I ±0.1 I
Patient with radium poisoning in front of apparatus—observed	7.22 I ±0.1 I
Actual gramma-ray ionization due to radium in body	0.32 I ±0.14 I

Since our calibration shows that 1 microgram of radium evenly distributed in the body produces an ionization of 2.90 I in our apparatus, we have here a case where 0.32/2.90=0.11 micrograms (μg) radium is located in the body, as revealed by its gamma-ray effect.

The total radium burden of this person, however, is much greater, since part of the radon produced by the radium in the body is exhaled and with it its main source of gamma rays. This amount has to be determined by the direct measurement of the radon exhalation in breath samples taken from the same person (see Part II of this paper).

The correction for the wall effect absorption in the body of the patient can, however, be avoided entirely by having the patient screened from wall radiations.

This was done by erecting a solid wall of iron bars, 10.5 cm. thick, 110 cm. high and 82 cm. wide behind the patient. The apparatus is then placed at such a distance from this iron wall that the solid angle subtended by this wall, as seen from the center of the measuring apparatus, is greater than the one subtended by the body of the patient sitting at the standard distance of 35 cm. from the center of the ionization chamber.

This has the further advantage of reducing the background ionization by about 2 I.

Discussion. The accuracy of our gamma-ray method is obviously dependent on the actual amount of radium lodged in the body. For patients with a large radium burden (for instance, several micrograms) the gamma-ray test will be accurate within a few per cent. The upper limit of radium in a living person, measurable with our method, would be about 30 micrograms.

Persons who have a radium burden of less than 0.1 microgram have to be measured more carefully and the accuracy of the result will be determined by the constancy of the background. If the background does not change by more than ±0.1 I, the determination of 0.1 microgram in the body (0.29 I) is accurate within ±30 per cent.

For persons with only incipient radium poisoning (for instance, 0.02 microgram), the effect of gamma rays from the body is of the same order of magnitude as the possible change in the background ionization and, in such cases, one has to carry out repeated

gamma-ray tests with and without the patient in order to be sure of a positive result.

Summarizing, we may say that our gamma-ray method *allows the detection of the tolerance dose of radium in the body (0.1 microgram) with sufficient accuracy within one hour, and of even smaller quantities if several tests are made in succession.* The method is superior to quantum counter measurements in its sensitivity, its readiness and constancy of its calibration. Moreover, it works under all atmospheric conditions.

II. DEVELOPMENT OF THE DIFFERENTIAL CHAMBER METHOD FOR ROUTINE MEASUREMENTS OF THE RADON CONTENT OF BREATH SAMPLES

Considerable progress has been made in the past few years in the technique of measurement of small quantities of radon. The use of proportional counters with amplifying circuits has the very definite advantage of eliminating the influence of particles of smaller specific ionization (cosmic rays, electrons); the observed pulses indicate only the rate of occurrence of alpha particles and the limitation of the method is given only by the magnitude of the background consisting of alpha particles from the wall of the counter.[7]

This type of apparatus is now being used for the measurement of very minute quantities of radon of rock specimens, breath samples, etc., in a few laboratories, as for instance, the National Bureau of Standards in Washington and the Massachusetts Institute of Technology.

They require elaborate precautions and constant attention by well trained personnel.

If we want to get quick information on the radon content of expired air from persons employed in the radium industry, we can use a much simpler method which was devised by Hess in 1929 and has been used for radon determinations by his pupils Roessner,[8] Zupancic[9] and Cullen[10] in various fields.

Since the two chambers are kept at the same voltage but of opposite signs, the charge received by the electrometer is zero, if the background ionization of the chambers are approximately equal. Even cosmic-ray showers do not disturb the equilibrium provided that they hit both chambers.

Steinke and Schindler[11] used the same principle in the study of cosmic-ray variations and Evans[12] constructed a similar differential double chamber apparatus with a self-registering string electrometer for radon determinations.

For the purpose of routine measurements of the radon content of breath samples, we must be able to determine if the sample contains more or less than 1×10^{-12} curie/liter which is the tolerance limit accepted at present.[13] A method suitable for this need not be too accurate for smaller amounts, say 2.10^{-13} curie/liter, but it should be reliable for finding cases approaching or exceeding the tolerance limit.

While working with the differential chamber method on another problem,[14] certain possible simplifications were explored and incorporated in the set-up of the apparatus described below.

If a breath sample of, for instance, 2 liters of expired air containing 10^{-12} curie/liter is introduced in one of the two chambers, the ionization produced after the radioactive equilibrium is reached (i.e. after three hours) corresponds to the combined action of the alpha rays of radon, RaA and RaC. Therefore, $3.7\times10^{+10}\times3\times10^{-12}=11.1\times10^{-2}$ alpha particles are emitted per second. For a measuring period of about 1,000 seconds 110 alpha particles are emitted and the natural fluctuation of the current produced amounts to only ±10 per cent.

Therefore, for our purpose, short collection periods of fifteen minutes, repeated once or twice will be sufficient.

The actual saturation current produced per curie of radon plus RaA and RaC was regularly determined by using radium standard solutions of 10^{-11} and 10^{-10} g radium provided by the National Bureau

of Standards in Washington.

The capacitance of the apparatus was also determined with a Gerdion variable condenser loaned to us by the Department of Terrestrial Magnetism (Carnegie Institution), Washington, D. C. This was done because we wanted to have an independent check when using, at the same time, two independent differential chamber apparatus, one connected with a Wulf unifilar electrometer, the other one with a Lindemann electrometer. The latter was found superior because of the remarkable constancy of the zero point of the electrometer during changes of the room temperature. The capacitances of the two complete apparatus (two 4 liter brass chambers, connecting tubes and electrometer) were found to be about equal (20.87 and 21 cm. respectively). The sensitivities used were about 60–70 scale div./volt. The calibration with different radium standard solutions, repeated several times during the past two years, with different accumulation periods, agreed very well. This remarkable constancy of the sensitivity of the apparatus is of great advantage for routine measurements. On the average, 1 micromicrocurie (10^{-12} cm.) was found to give a drift of 4.87×10^{-5} volts/sec. Therefore, the electrometer needle will indicate a drift of about 30×10^{-3} volts in five minutes, if a 2 liter breath sample of 10^{-12} curie/liter is introduced. This corresponds to more than 2ScD deflection of the needle per five minutes.

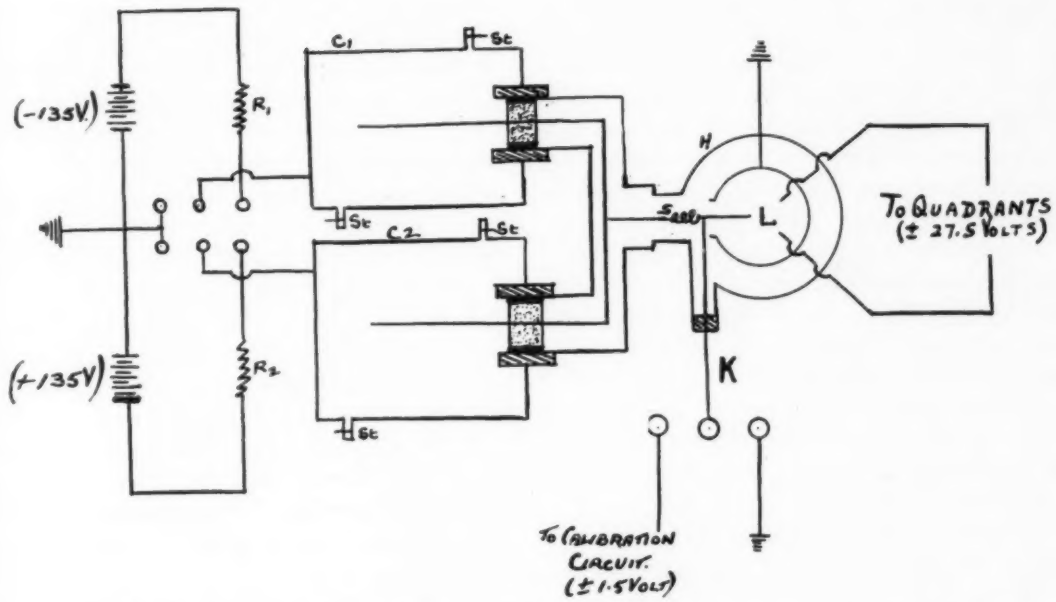
The two chambers were placed side by side on a wooden stand. The radon samples were always filled into the chamber which was connected to the +135 volt terminal. Therefore, the active deposit was collecting on the inner electrode of this chamber. Seven chambers of the same dimensions, 4 liter volume, were used and were easily replaceable after each measurement. One of the chambers was always used as compensating chamber, without radon filling.

The wiring of the whole apparatus is shown, in general, in Figures 3 and 4. Two panel boxes contained the batteries for the sweep potential on the chamber (plus or minus 135V), the quadrants of the electrometer and the circuit for the calibration of the electrometer. The complete assembly of the apparatus is shown in Figure 4. To the left is shown the high pressure tank with nitrogen and the manometer. The panel box to the left of the chambers contains the batteries for the sweep voltage (plus or minus 135V), the box to the right shows the voltmeter for the calibration.

The operation of the apparatus is very simple. First, all chambers are evacuated and then filled with dry, aged nitrogen at atmospheric pressure, from the high pressure tank (Fig. 4). Then, each of the chambers is tested with the same compensating chamber for the background. These tests take about twenty minutes for each chamber. Following this, each chamber is evacuated and connected through a 30 cm. long drying tube (containing P_2O_5 spread over the inner wall) with the spherical two liter flask containing the breath sample. After opening the stopcock the air from this flask streams into the drying tube and the ionization chamber. The other end of the sample flask is dipped into a jar filled with tap water and as soon as the stopcock of the flask (being under water) is opened, the outer atmospheric pressure forces the water in and pushes the remaining air into the drying tube and the ionization chamber. As soon as the sample flask is completely filled with water, all stopcocks are closed. Then, the ionization chamber is filled up to atmospheric pressure from the nitrogen tank, put on its stand and connected to the electrometer. If necessary, breath samples of an individual of about 4 liters each could be filled into the chamber. The time of filling is noted and three hours afterwards the measurements are performed, for each chamber. Tests of the sensitivity and of the zero point are made before and after each measurement.

When all readings are completed, the chambers are evacuated and refilled with inactive nitrogen to have them ready for the tests on the next day.

In more than two years of operation, we



C_1, C_2 - Ionization Chambers. S - Detachable spring connection to Electrometer
L - Lindemann Electrometer. H - Outer Housing of Electrometer.
R_1, R_2 - Resistors. K - Contactor for grounding or calibration.
St - Stop cocks for evacuating or filling the chambers.

Fig. 3. Differential double chamber for radon measurements.

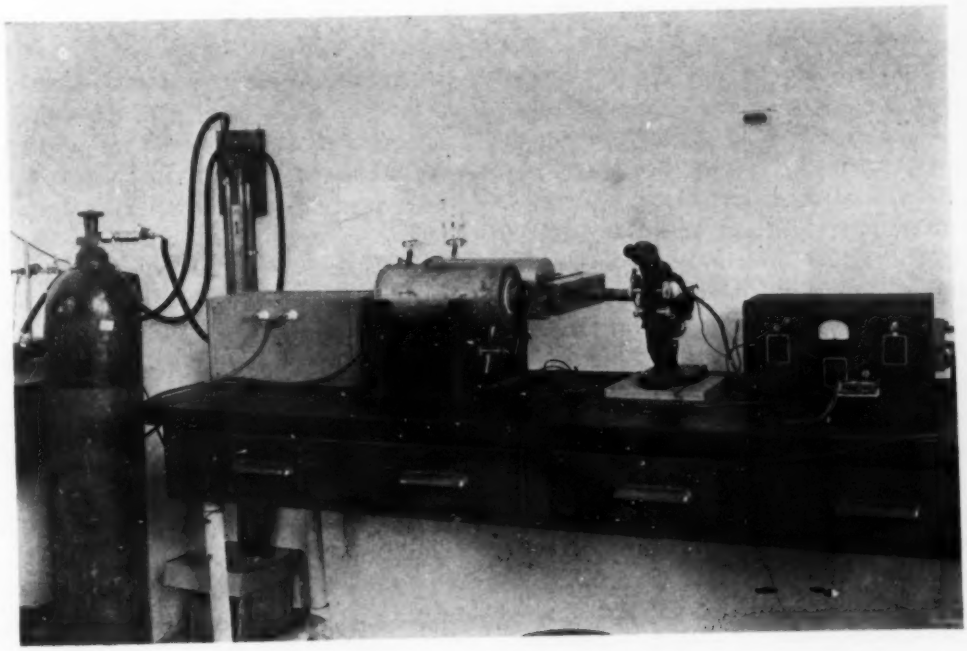


Fig. 4

did not experience any trouble from faulty insulation or from contamination of the chambers. It is important to keep the chambers on voltage during the three hours from the time of the filling to the time of the measurements (to insure a definite distribution of RaA, RaB, RaC in each chamber).

Another important feature is the use of a double housing of the Lindemann electrometer with a drying tube constantly supplied with fresh metallic sodium. This insures perfect insulation of the electrometer and of the ionization chamber even during hot and humid summer weather. The double housing of the electrometer prevents also sudden variations of the temperature from influencing the electrometer system. The contactor key (used for grounding the electrometer fiber or for testing the sensitivity) is directly built into the outer housing and is rigid enough to avoid any lever action on the rod leading to the fiber system.

We were using a loose cotton filter in a glass tube between the sample flask and the drying tube in order to trap large ions which may be produced during the rush of water into the sample tube. Later, we omitted this because Nolan and Fahy[15] found that small amounts of radon are absorbed by cotton.

We tested this, however, calibrating the apparatus with the radium standard solution and found no difference in the results with or without the use of a loose cotton wad in the filling line. It is probable that an effect of this sort will be noticeable only if a very densely packed cotton filter is used.

The apparatus described here was tested repeatedly by introducing blank samples (sample flask filled with inactive nitrogen or atmospheric air) and we found that it always was working satisfactorily.

Concentrations of radon from 0.2×10^{-12} curie/liter up can be determined with sufficient accuracy and this accuracy is, of course, greatly increased for concentrations of 10^{-12} curie/liter and above.

Our experience with a hundred or more actual tests of breath samples from employees of commercial concerns shows that tests on the same person, repeated within short intervals of time, check remarkable well, within ± 20 per cent in most cases.

The agreement is less satisfactory if different procedures are used in taking the breath sample from each individual. In our experience, the use of rubber or neoprene balloons in collecting samples is certainly not recommended. Part of the exhaled radon is absorbed by the material of the balloon if the sample is not immediately transferred into a glass flask. We, therefore, following the method used by Evans, collected the samples by requiring the individual to breathe directly through a flask with two stopcocks.

It is, of course, not advisable to use the apparatus in buildings which are not free from radioactive contamination.

Samples from persons should also always be taken in uncontaminated rooms, preferably with all windows open, since it is well known that certain building materials exhale enough radon to produce concentrations of 10^{-12} curie per liter of room air with all windows closed.

We also found that if breath samples are taken in such an atmosphere, the air exhaled from persons is in most cases less radioactive than the room air. It seems that part of the radon inhaled in the breathing process is absorbed in the lungs and therefore the exhaled air contains less radon.

Comparison of the Radon in the Breath with Radium Content of the Body and the Determination of the Total Radium Content. The radon exhaled with the breath indicates the presence of radium in the lungs and in the blood stream. Evans has shown that 1×10^{-12} curies of radium in the breath corresponds to 0.09 microgram in the body. In addition to this, radium affixed to the bones and other tissues and organs does not give off its radon and is therefore detectable only by its gamma radiation (which in its main part comes from RaB and RaC) including RaC″.

The total amount in the body is found, therefore, by adding the two quantities of radium determined (*a*) by examination of the breath sample, and (*b*) by gamma-ray measurements.

The ratio of the two components varies in individual cases. Evans[1] found that on the average 45 per cent of the radium in the body is detected as radium in the breath and 55 per cent is detected by the gamma rays from the body. In about 20 per cent of the cases, he found significant variations of these figures.

Our experience corroborates, on the whole, these results. Selecting a group of 26 radium workers, we found that in 16 cases, the ratio (p) of radium equivalent of breath: radium in body (gamma-ray method) was between 0.4 and 1.5 (average 0.92) which is not very different from Evans' figure $45/55 = 0.82$. In 10 cases, very different ratios ranging from $p = 0.09$ to 13.3 were found (average 3.3). The total average for p computed from all 26 cases is 2.1, much higher than Evans' mean value. This would indicate that the average exhalation in our group is about twice the equivalent of the mean radium content of the body.

These distribution figures may well be very different, depending on the time of exposure and the difference in the chemistry of the body of individuals.

We measured one individual who presumably acquired radium in his body twenty years ago in the Middle West, and the ratio of the radioactivity of the breath to the radioactivity of the body was practically unchanged for a period of nearly one year, our findings showing $p = 0.30$, 0.47, 0.46, 0.63, 0.50. The situation may be very different for recently acquired quantities of radium.

Responsible firms in the radium and luminous material industry have now adopted the plan of having all of their employees checked for radon in the expired air twice a year. For employees exposed to greater hazards (technicians and chemists) expired air samples should be tested every three months and, in all cases, where more than the tolerance limit is found, a check of the radium content of the body should be made using the gamma-ray method (as described in the first part of this paper or by using quantum counter).

If such a procedure is adopted and adhered to strictly, all cases of incipient radium poisoning can be detected in time and proper measures taken by the medical authorities. Several states have put in force such regulations for the protection of workers in the radium industry.

SUMMARY

1. An integrating gamma-ray method was worked out which allows for determining quantities of radium of the order of 0.1 microgram in the body with a fair degree of accuracy within one hour. Amounts of only 0.03 microgram can be estimated within plus or minus 0.01 microgram. Thus, it is possible to detect incipient radium poisoning before the tolerance limit (set at present as 0.1 microgram) is acquired.

2. The double chamber differential method of determining the radon content of expired air samples from persons employed in the radium industry was improved and simplified for routine work. Concentrations of much less than 1 micromicrocurie per liter of exhaled air can be measured within a few hours.

Practical experience with this method at Fordham University during the past two years is reported.

Physics Department
Fordham University
New York 58, N. Y.

REFERENCES

1. Evans, R. D. Radium poisoning. II. Quantitative determination of the radium content and radium elimination rate of living persons. Am. J. Roentgenol. & Rad. Therapy, 1937, *37*, 368–378. Protection of radium dial workers and radiologists from injury by radium. *J. Indust. Hyg. & Toxicol.*, 1943, *25*, 253–269.

2. Hess, V. F. *Tr. Am. Geophys. Union*, 1946, *27*, No. 5, pp. 670–676. A more detailed description

will be given by O. H. Gish and K. L. Sherman in *Terr. Magn. & Alm. El.*

3. WILSON, C. T. R. *Proc. Camb. Phil. Soc.*, 1900, *11*, 32. *Proc. Roy. Soc., London*, 1901, *48*, 151.
4. COMPTON, A. H., WOLLAN, E. O., and BENNETT, R. D. *Rev. Sci. Instruments*, 1934, *5*, 420.
5. SCHLUNDT, H., BARKER, H. H., and FLINN, F. B. Detection and estimation of radium and mesothorium in living persons. I. AM. J. ROENTGENOL. & RAD. THERAPY, 1929, *21*, 345–354.

   SCHLUNDT, H., and FAILLA, G. Detection and estimation of radium in living persons. III. Normal elimination of radium. AM. J. ROENTGENOL. & RAD. THERAPY, 1931, *26*, 265–271.

   SCHLUNDT, H., NERANCY, J. T., and MORRIS, J. P. Detection and estimation of radium in living persons. IV. Retention of soluble radium salts administered intravenously. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *30*, 515–522.
6. HESS, V. F., and BALLING, EVA. *Tr. Am. Geophys. Union*, 1945, *26*, 238.
7. BROWN, S. C., ELLIOTT, L. G., and EVANS, R. D. *Rev. Sci. Instruments*, 1942, *13*, 197.
8. ROESSNER, H. Thesis (Junsbruck, 1931). *Mineral. Petrograph. Mitteil.*, 1933, *44*, 495.
9. ZUPANCIC, R. *Terr. Magn.*, 1934, *39*, 33.
10. CULLEN, T. L. *Terr. Magn.*, 1946, *49*, 37.
11. STEINKE, E., and SCHINDLER, H. *Naturwiss.*, 1932, *20*, 15.
12. EVANS, R. D. Apparatus for the determination of minute quantities of radium, radon and thoron in solids, liquids and gases. *Rev. Sci. Instruments*, 1935, *6*, 99–112.
13. National Bureau of Standards, Washington, D. C., Handbook H27 (1941).
14. HESS, V. F. *Tr. Am. Ceophys. Union* (Sect. of Hydrology), 1943, p. 587.
15. NOLAN, P. J., and FAHY, E. F. *Proc. Roy. Irish Acad.*, 1945, *50*, A, 233.

Vol. 57, No. 1

FUNDAMENTAL PRINCIPLES IN THE APPLICATION OF CINEROENTGENOGRAPHY AS AN AUXILIARY METHOD TO ROENTGEN DIAGNOSIS

By JOSÉ MORETZSOHN DE CASTRO, M.D.
Head of the Radiological Department—Gastroenterological Division "Dr. Levy Sodré"—Santa Casa de Misericordia; Founder of the Brasilian Institute of Clinical Cineradiography
SÃO PAULO, BRAZIL

THE idea and the first realization of taking moving pictures of the fluorescent screen for the documenting of roentgenoscopy—so-called indirect cineroentgenography—belong to Russell Reynolds of England. Later, several authors devoted themselves to the subject, but, strange to say, cineroentgenography has not yet been applied to daily practice.

My personal experience in cineroentgenography dates back to 1939, when my experimental work with Mr. José Jany began in São Paulo, Brazil. Several of the results of these experiments have been published both in Brazil and in other countries.

In this present work I shall not describe the apparatus devised by Jany and which was used in the experiments; those who wish to obtain the detailed description of this apparatus will find it in the articles already published.

The technical details solved by the Jany apparatus and which permit the practical use of cineroentgenography in daily clinical practice are as follows:

(1) It is an accessory for cineroentgenography, adaptable to common roentgen installations without any change being effected in the transformer or in the cooling system of the tube.

(2) Used as an accessory, it also permits the roentgenographing of the fluorescent screen in any position that the roentgen table may be.

(3) Owing to the use of a bellows, roentgenograms may be taken in an illuminated room.

(4) A perfect system of synchronization and perfect visibility of roentgenoscopy through the use of a telescopic viewfinder.

We have taken nearly 1,700 cineroentgenograms with the Jany apparatus, using 16 mm. cinefilms and more recently, with the improvement of the apparatus, we have been able to use 35 mm. films, preferring the latter.

The superiority of the 35 mm. films is undeniable, for the following reasons: (*a*) a larger sized image which gives better roentgenoscopic details; (*b*) sharper images both in continuous projection and fixed projection, when the film is projected frame by frame; (*c*) little difference between the price of the 16 and 35 mm. film; (*d*) greater facility in finding a larger variety of films of different emulsions on the market; (*e*) selected material may be used from the roentgenologist's files for the production of cultural films; (*f*) the continuous projector may be dispensed with because the images may be quite well observed through a viewing box which is always at hand on the roentgenologist's table; (*g*) the reduction of the 35 mm. negative to 16 mm. positive is undoubtedly superior to the enlargement of the 16 mm. to 35 mm.

Not only are the technical problems practically solved by Jany, but so are the problems pertaining to the photocinematographic laboratory, which in themselves represent one of the most important features for obtaining the final result—good roentgenograms, good copies and good enlargements. This part has been in charge of Mr. Harry C. R. Tuff, who has collaborated with us since the beginning of our experiments. Thanks to the close cooperation between the technicians and the roentgenologist, we have achieved good results.

Clinical cineroentgenography is a method of roentgen examination and therefore does not constitute an invention. Using an apparatus devised in Brazil in accordance with our technique for roentgen examination, we have only introduced the "method" as it should be applied in order to be of practical use.

Individual capacity of observation—in roentgenology—is limited to our sense of vision, which gives us only a subjective idea of the kinetics of the organ.

If we wish to *fix* our roentgenoscopic findings, we are obliged to employ the cinematography of the fluorescent screen as the only *technigraphical* means at hand, at least for the present, for the serial documentation of the movements observed during the examination.

The motion of an organ—although it does not express the totality of its vital functions—nevertheless represents the externalization of the motor function, the only one which may be registered on cinematographic film for later observation.

Beginning with the simplest reasoning and leading to the most complex, we find four advantages which indirect cineroentgenography has brought to clinical roentgenology:

1. With the advent of the "Jany" apparatus we have been able to produce sharp roentgenograms of the fluorescent screen, thus documenting the roentgenoscopy.

2. The projection of the cinematographic film permits better subjective observation, because one may use slow projection and slow motion.

3. Fixed projection of each frame in turn allows one to make a still better analysis of the movement, with detailed study of the variations of the successive roentgenoscopic images. This form of analysis gives much more clinical data than the continuous projection, but it still continues to be an observation of the *subjective* form—personal or collective—according to the number of observers present.

4. By tracing the outlines of the organs observed in the fixed projection on graph paper (in millimeter squares), one may analyze the movement *objectively* and *mathematically*, studying the alterations of the forms of the organs, not only in the bidimensional fluorographic plane, but at the same time introducing the exposure time of the images (frames) as a new chronometric dimension for the evaluation of the motor function.

To this fourth form of analysis of the cineroentgenographic film, we have given the name "roentgenological chronocinegraphy," and we have defined it as being "the roentgenological documentation of the movement of an organ in relation to time."

The diagram of the roentgenological kinetics obtained by the tracing of the various consecutive images has been denominated by us as being "the chronographic inscription in coordinated lines of the phrase in motion" (lit.).

In clinical roentgenology we are not interested in the filming of each and every movement which we observe in roentgenoscopy, because many of them may be sufficiently well analyzed on the fluorescent screen alone.

The roentgenologist should make use of cineroentgenography only when:

(*a*) He does not consider the movement observed in roentgenoscopy sufficient for diagnosis;

(*b*) When the motor phenomenon which he wishes to analyze is extremely rapid, as, for example, in the case of a duodenal dyskinesia, a pharyngeo-esophageal disorder, etc.

(*c*) When, owing to divers circumstances, we cannot repeat in roentgenoscopy as often as we may wish for a perfect analysis, the phase in motion which is of interest; for example, the observation of the contrast mass in the stomach for the determination of the index of tonicity of the stomach walls.

(*d*) When it is necessary to study the functional interdependence of two or more organs situated outside of this visual field of observation. Example: the synchronous analysis of the influence of various factors,

thoracic, cardiologic and diaphragmatic, which act upon a pulmonary cavity.

Once the film is made with the functional pathological cycle registered, one has only to choose the best method of analysis applied to the case under study.

The functional roentgen diagnosis should —according to present knowledge of physiology and physiopathology—be sufficient to explain the predominant objective symptom indicated by the patient.

Any functional disorder, even if only initial, rarely passes unperceived by the patient and will be evidenced by subjective symptoms, more or less accentuated, in accordance with its extension and localization.

The roentgenologist must localize and fix this disorder and, in his capacity as a technician and doctor, analyze and clinically interpret it.

Before taking a roentgenogram of any organ, the roentgenologist should consider two fundamental problems: one of a technical order and the other of a medical-technical order: (1) how to take the roentgenogram for the selection of the pathological functional cycle; (2) the duration and the velocity of the movement.

(1) In the selection for the roentgenographing of the functional cycle, the roentgenologist must consider the following: (*a*) that its duration is known beforehand; (*b*) that its localization is or is not known beforehand.

(*a*) When making a film of the kinetics of the heart, we know beforehand the state of the patient's pulse. Thus we know, beforehand, the total duration of the motor phenomenon which we wish to register. It is sufficient to film the fluorescent screen, with the largest possible number of images, so as to disassociate this movement for later interpretation. I believe that after taking a roentgenogram of the heart one should always make a roentgen chronocinegram of the same, so as to obtain a better analysis, and also to provide the clinician with some documentary proof.

(*b*) A patient with marked dysphagia incidentally indicates subjectively the segment of the esophagus to be filmed; thus, the localization of the disorder becomes simple. Take a film of the region indicated and control the filming by means of the tele-view finder of the "Jany" apparatus.

A patient who had undergone an appendectomy and who felt pains in the right iliac area some minutes after partaking of food would thus indicate the proper region to be studied (ascending colon, terminal portion of the ileum).

It is necessary to register the functional interdependence of the painful phenomenon by means of the gastric filling.

Then we apply a simple technique for the location of the functional trouble and explanation of the subjective phenomenon—pain.

We ask the patient to take a barium mixture nine hours before the examination; we verify by means of roentgenoscopy that the last ileal loop—the cecum and the ascending colon—are visualized. Next he is given some food to eat which habitually provokes pain.

Three cineroentgenographic images (frames) every three minutes before, during, and after swallowing of the food were sufficient to register all of the functional alteration which caused post-prandial pain. We register the moment at which the pain appears and the instant in which the symptom begins decreasing as well as when it finally disappears.

The diagram of the film and the curve of the development of the pain gives us sufficient material for the interpretation of the etiology of the post-prandial pain.

In this case the patient was suffering from a disturbance of the sphincter, with the consequent progressive increase of the intracecal pressure, coinciding with the irritation of the pain at that instant.

(2) Duration and velocity of the movement. We will consider two kinds of movement—voluntary and involuntary.

The filming of organs which have voluntary movement have little clinical interest. In the cases in which there may be interest

in taking films of these movements one should reduce the velocity of the movement as much as possible in order to obtain greater detail on the film.

FIG. 1. Continuous film of the esophagus taken at the speed of 16 frames per second. Patient in prone position. Each frame exposed at 1/32 second with an interval of 1/32 second between each. The transit was extremely low, this being a case of avitaminosis of the B complex type.

The involuntary movements may be subdivided into two classes: (*a*) movements of short duration: ordinary filming, trying always to obtain the maximum of frames (images) for better registering; (*b*) movements of long duration: as one may take films for hours at a time, they should be divided into short series of three or more frames each, always using chronometrical control of the intervals between the various series of frames in order to obtain a perfect analysis later.

As an example may be mentioned the

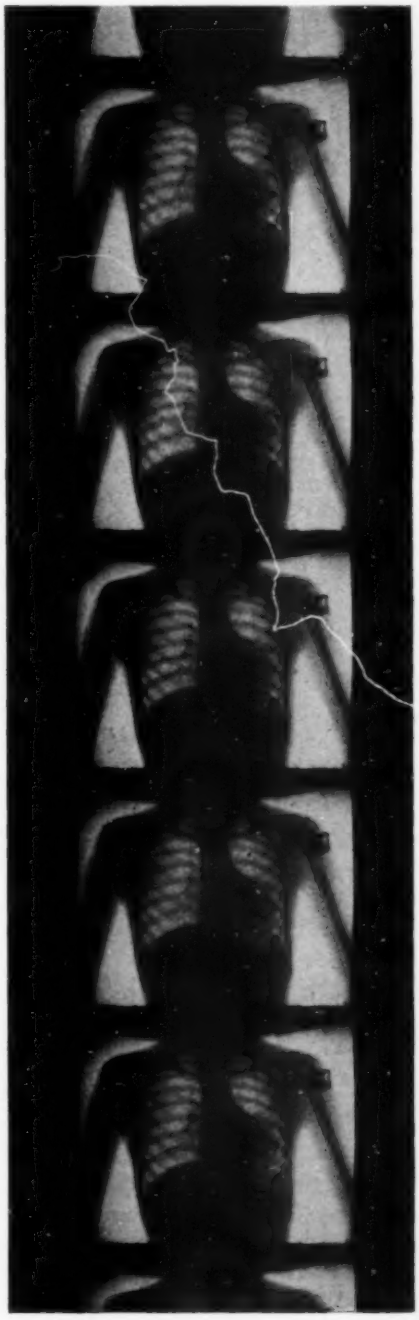


FIG. 2. Enlargement of 16 mm. fine grain film (previously hypersensitized) taken at 24 frames per second. Case of compensated congenital cardiopathy.

gastroenterocolic transit cycle. In our clinic we proceed as follows: (1) Continuous cinematographic registration of the passage by the cardia until the caudal pole is reached (for the determination of the gastric tonus). (2) Test of the total gastric filling—continuous filming until the point of distention of the antrum is reached. (3) The three frames are taken every three minutes during three hours, intercalating ordinary roentgenography when judged necessary.

The later study should then be made by the projection of the film, frame by frame, and also through the tracing of the phase which may be of interest.

Each one of the points which I have mentioned here would require a special chapter owing to their importance. That constitutes a task which I am at present undertaking and which I hope to publish shortly from my personal observations.

Herewith in the illustrations are presented some cases to demonstrate that what has been said above has been realized practically and that nothing has been based upon theory alone (Fig. 1–8).

As has already been stated, in not all of the cases is the simple projection frame by frame sufficient. This is shown by the following case:

Case 1 (No. 9758). N. D., male, suffered from chronic ulcer of the duodenum for nine years (roentgen diagnosis). During the last three years the patient had not suffered any painful epigastric crisis. Six months before the present examination by the author, he felt acute pain in the right iliac region which radiated to the corresponding member. Since then during these appendicular crises there was an increase of the painful epigastric symptoms.

Von Bergman had already mentioned and we had occasion to confirm the truth of his statements, together with Dr. Waldeloyr Chagas de Oliveira (*Valor da fisio-radiologia na propedeutica cirurgica dos colos*, Brazil, 1943), that in cases of appendicitis, as a rule, one observed hypertonia of the cecocolic sphincter.

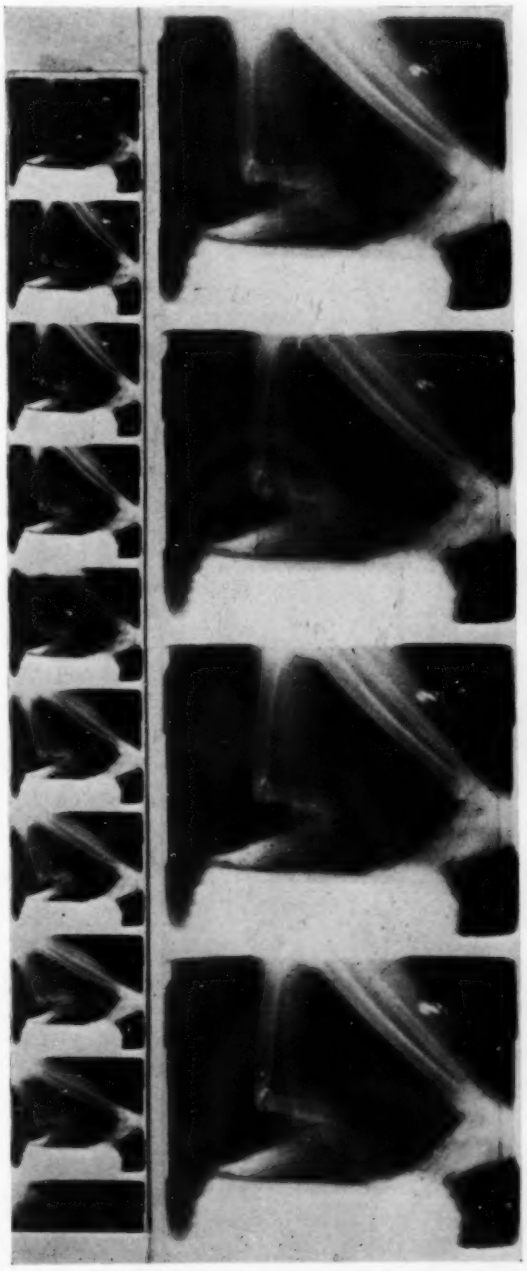


Fig. 3. Congenital pseudarthrosis. Continuous film film taken at 16 frames per second on 35 mm. film. This film was made for filing, for the ultimate purpose of compiling cultural and didactical films of general interest. One contact print and one enlargement.

On the other hand, the influence of the presence of the internal ambient of the cecum upon the progression of the ileal contents is well known.

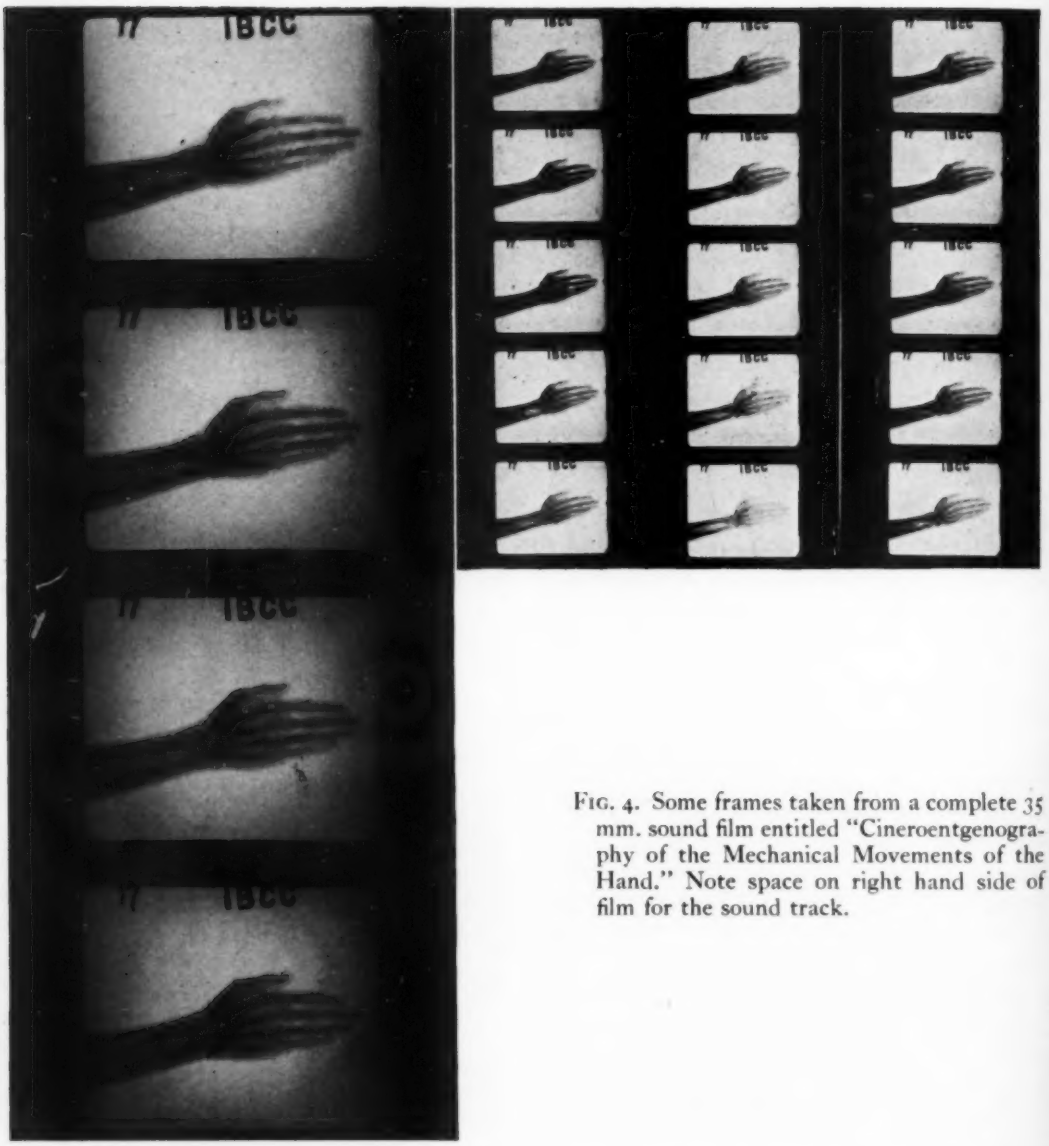


FIG. 4. Some frames taken from a complete 35 mm. sound film entitled "Cineroentgenography of the Mechanical Movements of the Hand." Note space on right hand side of film for the sound track.

In 1942, together with Fernandes Pontes, I verified that the increase of the ileal pressure provokes spasmodic contractions of the gastric antrum. At that time I considered the reflex observed to be similar to that of Hirsch, but inverted, and therefore purely neurovegetative. We now have the impression that the phenomenon is more complex, principally from the work of Ivy.

The subjective gastric sensations, during an appendicular crisis are all very well known.

In order to document the reason for the gastric symptomatology presented by the patient during the appendicular crisis, I proceeded as follows:

Nine hours before the roentgen examination the patient was given by mouth 150 grams of barium sulfate diluted in an equal quantity of water. The patient was then placed in the "Jany" apparatus for cineroentgenography and approximately 20 frames were taken at pre-established exposure and interval times. During the filming, roentgenoscopy was maintained by means of the teleview finder. These 20

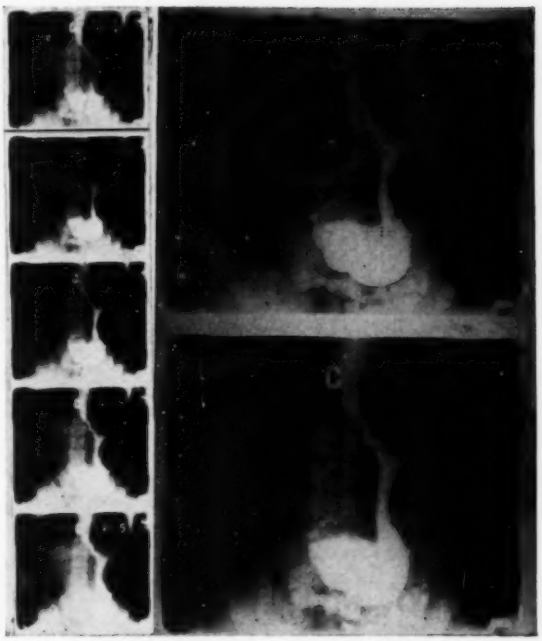


Fig. 5. Direct contact copy of 5 frames documenting the passage of the contrast medium through the cardia where, slightly below, one may observe a pseudo-diverticulum. Also note the deformity of the duodenal cap. Observe the hypotonia of the esophagus and the stomach. This is a case of chronic ulcer of the duodenum with hypokinetic syndrome through lack of the B vitamin complex.

frames were sufficient to register the total filling of the stomach and duodenal cap. When the contrast medium passed the upper knee of the duodenum, the filming was stopped because the second portion was then behind the cap.

Following this an ordinary roentgenoscopy and serial roentgenograms of the duodenal cap with compression were made in order to obtain an outline of the mucosa. Next, 1 cc. of beta-hypofamina was injected intramuscularly into the patient and cineroentgenography was continued of the provoked peristalsis, according to the technique of Garcia Capurro, combined with that of Chagas de Oliveira. The next step was roentgen examination of the appendix,

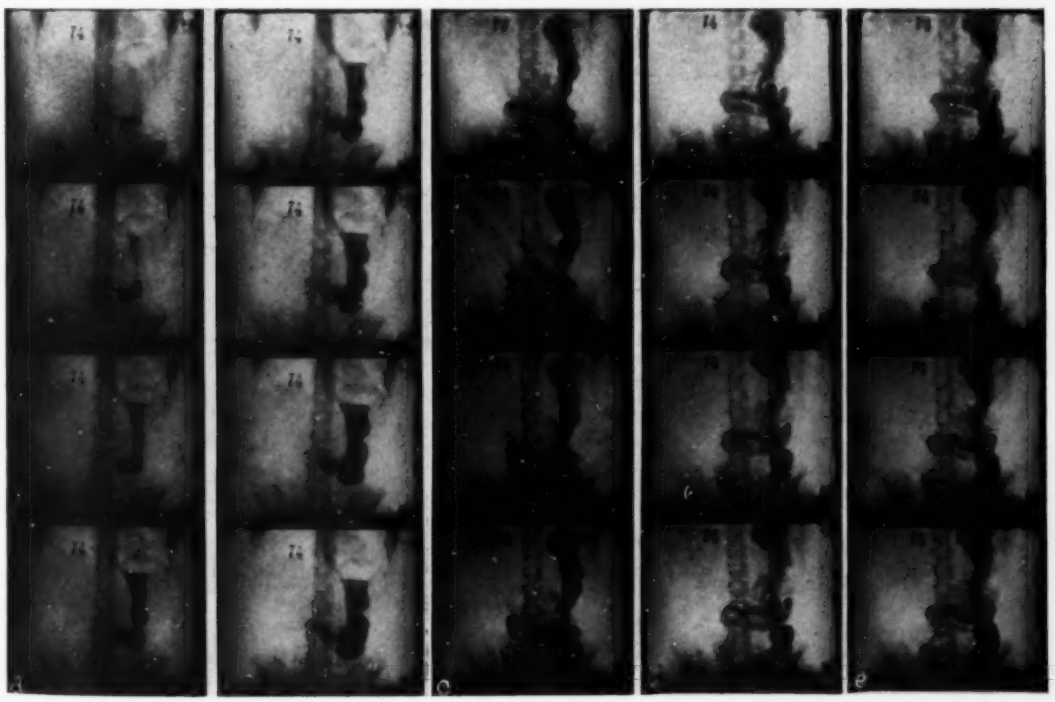


Fig. 6. *a*, *b*, *c*, *d*, *e*, aspects of jejunogastroduodenal transit. *a* and *b*, filling of the stomach, some enlarged frames. Patient in orthostatic position. *c*, *d* and *e*, documentation of the movement of rhythmic segmentations and progression of the jejunum. Note the high situation of the stomach, a result of the position of the patient (prone position).

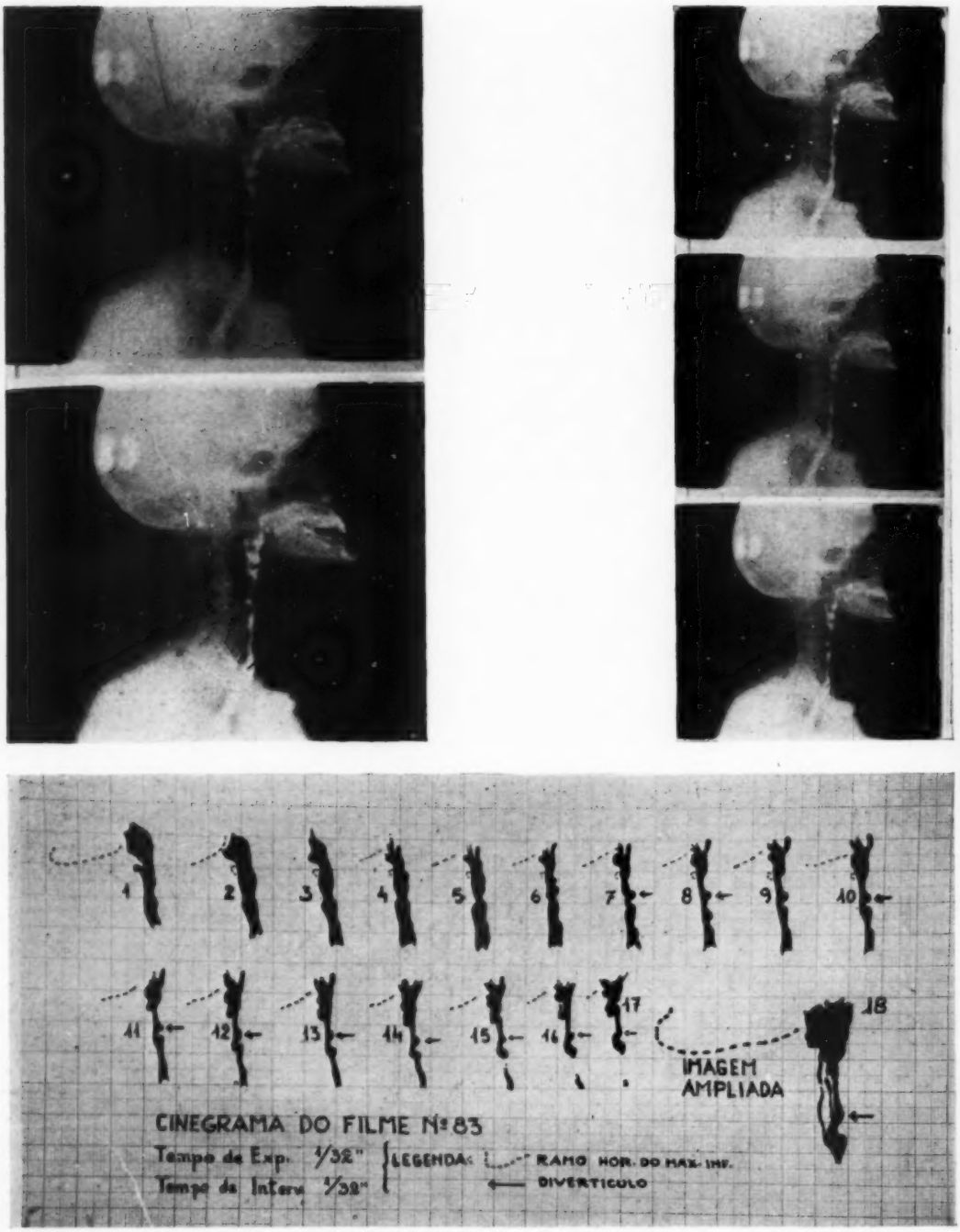


FIG. 7. Pharyngo-esophageal diverticula (Zenker). Verified by endoscopic examination (case of Dr. Plinio Mattos Barretto). Sixteen frames during one second where the diverticulum is evident in only 8 frames. By the ordinary methods of roentgen examination this diverticulum could not be diagnosed and even after knowing of its existence we were unable to see it during roentgenoscopy. The chronocinegram shows that it is only a hernia of the mucosa and only visible in the phases of pharyngeal hypertension. The maximum extension was reached at the eighth frame, corresponding in the diagram to 2 millimeters. The maximum width of its base is 3.5 mm. (tenth frame). These measurements are relative and if it is wished to obtain the natural size, it is only a question of ascertaining the scale of the diagram.

Fig. 8. Enlargement of a 35 mm. film made at the speed of 8 frames per second, using a thick barium paste. Notwithstanding this, the descent of the barium through the pharynx was very rapid. In 4 frames one may see a pseudo-diverticulum in the pharyngo-esophageal region (Zenker). The patient was a female, aged forty, who during one year suffered from high dysphagia, the cause of which could not be identified until this cineroentgenographic examination was made. As in the previous case (Fig. 7) even knowing of the existence of this lesion, we were unable to note its presence during roentgenoscopy.

The cases shown in Figures 7 and 8 are sufficient to prove what I have heretofore affirmed when I mentioned that certain phenomena which pass rapidly before our vision during roentgenoscopy and projection on the cine-screen, go unperceived and therefore necessitate the disassociation of the film, frame by frame, with the incidental tracings and chronocinegrams to demonstrate and confirm their presence and functional alterations, in relation to the time of duration of the phenomena.

Fig. 9

Fig. 10

Fig. 11

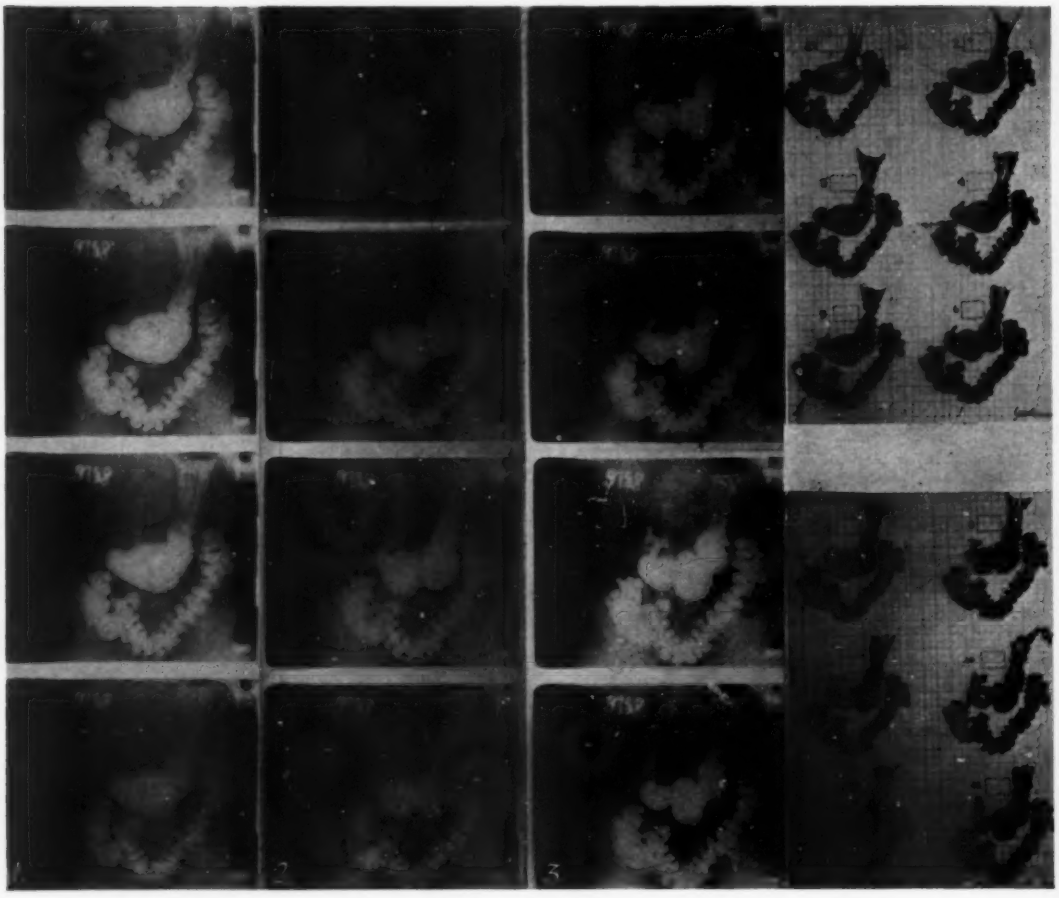


FIG. 12, *1*, *2*, *3*, *4*, *5*.

which showed all of the classical signs of appendicitis including pain on palpation.

After the cinefilm and roentgenograms were developed I proceeded with the technique for the taking of the chronocinegram, etc.

In order to make the chronocinegram, it is necessary to have a special table equipped with technical requisites which facilitate the tracing work. So far, we have been unable to find a definite table suitable for this work, but I shall here explain how we drew up the chronocinegrams which illustrate this work.

Figure 9 shows a still projector (S.V.E.) in which one may insert a roll of film and project it frame by frame onto a mirror set at an angle of 45 degrees in relation to the optical center of the projector objective. Thus the image is reflected down on to the table on which the graph paper is placed. The outlines of the images are then traced on this paper (Fig. 10 and 11).

The optical data of the objective being known factors, as is also the distance at which the film is projected, it is quite easy to determine the exact natural size of the roentgen image. It is sufficient to make the common optical calculations and relate them to the distance at which the film was taken, together with the distance between the roentgen tube and fluorescent screen, all of which data are easily obtained from the Jany apparatus.

In the present case, N. L. 9758, (Figures 12, *1*, 12, *2*, and 12, *3*), twelve images were traced, i.e., from the gastric filling to the

passage of the contrast medium through the pylorus, the filling of the duodenal cap and the passage through the upper knee of the duodenum. From these images, using the technique which we have already explained, we obtained the chronocinegram (Fig. 12, *4* and *5*) from which the following conclusions may be drawn:

The images *1*, *2*, *3* and *4* show an accentuated and progressive contraction of the antrum, the maximum pressure of which is attained in image *4*. One may also note that in this image (*4*) the intracecal pressure reached its maximum, having the Hirsch sphincter completely contracted. In the following images there are no particular alterations of the intracecal pressure which progressively returns to its former state, but after having transposed the pylorus and filled the superior recess (image *7*), the inferior recess being contracted, a new contraction of the entire cecum takes place at the instant in which the contraction of the middle part of the duodenal cap, together with a heavy contracting wave of the pre-antrum, cause an increase of the intrapyloric pressure and of the base of the duodenal cap (image *8*).

Modifications of the haustra of the transverse colon and of the splenic angle may also be noted.

The principal fact registered is the filling of the upper recess of the duodenal cap before that of the lower recess, thus documenting the spastic contraction of the latter which only fills up, three images before the first is fully distended (images *7*, *8*, *9* and *10*).

The hypertonia of the fiber at the lower base of the duodenal cap may be due to the duodenal ulcer, but at the same time it may be due to a motor reflex which begins at the cecum and appendix, and, by way of the parasympathic and neurohormonal, reaches the antrum and duodenal cap. The patient will undergo an appendectomy.

Later on, a new chronocinegram obtained under identical conditions will inform us as to the functional alterations then existing in the duodenal cap after having excluded one excito-motor reflex factor of the antrum, namely the appendix.

REFERENCES

1. Alvarez, W. C. An Introduction to Gastro-Enterology. Third Edition. Paul B. Hoeber, New York, 1940, p. 623.
2. Barclay, A. E. The Digestive Tract. Second edition. University Press, Cambridge, 1937.
3. Barclay, A. E. Direct x-ray cinematography with preliminary note of nature of non-propulsive movements of large intestine. *Brit. J. Radiol.*, 1935, *8*, 652–657.
4. Barclay, A. E. Normal mechanism of swallowing. *Op. cit.*, p. 138.
5. Castex, M. R., Mazzei, E. S., and Malenchini, M. Roentgencinematografia (con técnica de Abreu). *Arch. argent. de enferm. d. ap. respir. y tuberc.*, 1941, *9*, 300–306.
6. Castex, M. R., Mazzei, E. S., and Malenchini, M. Broncocinematografia. *Arch. argent. de enferm. d. ap. respir. y tuberc.*, 1941, *9*, 307–311.
7. Clark, K. C. Positioning in Radiography. C. V. Mosby Co., St. Louis, 1939.
8. de Abreu, M. Técnica de la roentgencinematografía. *Arch. argent. de enferm. d. ap. respir. y tuberc.*, 1941, *9*, 283–299. Also, *Día méd.*, 1941, *13*, 896–897; 908–909.
9. Dariaux and Djian. Solution pratique de radioscopie cinématographique par le procédé du docteur Georges Djian. *Bull. Soc. de radiol. méd. de France*, 1935, *23*, 344–346.
10. Dessauer, F. Ueber Kinematographie mit Röntgenstrahlen. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1937, *56*, 126–131.
11. Files, G. W. Factores que influyen en la nitidez del detalle o deficion. *Radiologie y fisioterapia*, 1940, *7*, 251.
12. Graf, H. Technische Grundlagen der indirekten Röntgenkinematographie. *Fortschr. a. d. Geb. d. Röntgenstrahlen* (Kongresshft), 1937, *56*, 87–90.
13. Grishman, A., Steinberg, M. F., and Sussman, M. L. Contrast roentgen visualization of coarctation of aorta. *Am. Heart J.*, 1941, *21*, 365–370.
14. Groedel, and Franke, H. Ueber technische Fortschritte der direkten Röntgenkinematographie. *Fortschr. a. d. Geb. d. Röntgenstrahlen* (Kongresshft), 1933, *48*, 65–67.
15. Janker, R. Roentgen cinematography. Am. J. Roentgenol. & Rad. Therapy, 1936, *36*, 384–390.
16. Janker, R. Leuchtschirmphotographie Röntgenreihenuntersuchung: Die Photographie des Leuchtschirmbildes: Eine Methode der Röntgenreihenuntersuchung. J. A. Barth, Leipzig, 1938.

17. KAESTLE, C. Zur Geschichte der Röntgenkinematographie (Bioröntgenographie) und ihrer Leistungen. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1937, *56*, 144–148.

18. KAWAISHI, K. Studies on roentgen cinematography of internal organs and circulation of the blood of the human body. AM. J. ROENTGENOL. & RAD. THERAPY, 1938, *40*, 913–921.

19. LOMON, and COMANDON. Radiocinematographie par la photographie des écrans intensificateurs. *Presse méd.*, 1911, *29*, 359.

20. LUBOSHEZ, B. E. Cineradiografia. *Radiol. med.*, 1931, *18*, 450–457.

21. MORETZSOHN DE CASTRO, J. A cineradigrafia clínica. *Anais do 2º Congresso Nacional de Tuberculose*, São Paulo, 1941, *3*, 409.

22. MORETZSOHN DE CASTRO, J., and JANY, J. O metodo brasileiro de cinerradiografia-clinica. *Rev. paulista de tisiol.*, 1942, *8*, 110–138. Also, *São Paulo méd.*, 1942, *2*, 207–224.

23. MORETZSOHN DE CASTRO, J., and JANY, J. O que nos promete a cineradiografia no estudo da fisiologie e da fisiopatologia do esófago. *Rev. de gastro-enterol. de São Paulo*, 1941, *4*, 43–48.

24. MORETZSOHN DE CASTRO, J., and LOCCHI, R. Mecanismo de fechamento da faringe nasal na deglutição, á luz da dissecçao e da cineradiografia. *Rev. sudamer. de morfolog.*, 1943, *1*, 44–62.

25. MORETZSOHN DE CASTRO, J. Cronocinegrafia radiológia; contribuição para o estudo do movimento dos órgãos em função do tempo. *Rev. brasil. de cir.*, 1944, *13*, 75–90.

26. MORETZSOHN DE CASTRO, J., and PONTES, J. F. Contribuição da cine-radiografia ao estudo da físio-radiologia do intestino delgado. *Rev. brazil. de cir.*, 1944, *13*, 91–102.

27. MORETZSOHN DE CASTRO, J., and DE MATTOS BARRETTO, P. Contribuição da cine-radiografia ao estudo das perturbações funcionais da deglutição. Nota prévia. *Rev. brasil. de cir.*, 1944, *13*, 103–106.

28. PANCOAST, H. K., PENDERGRASS, E. P., and SCHAEFFER, J. P. The Head and Neck in Roentgen Diagnosis. Charles C Thomas, Springfield, Illinois, 1940, p. 825.

29. REYNOLDS, R. J. Cineradiography. AM. J. ROENTGENOL. & RAD. THERAPY, 1935, *33*, 522–528.

30. REYNOLDS, R. J. Cineradiography. *Fortschr a. d. Geb. d. Röntgenstrahlen*, 1936, *53*, 602–606.

31. REYNOLDS, R. J. Cineradiography. *Brit. J. Radiol.*, 1934, *7*, 415–424.

32. REYNOLDS, R. Praktische Kineradiographie durch die indirekte Methode. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1937, *56*, 132–139.

33. ROSE, J. J. American Cinematographer Handbook and Reference Guide. 1935, p. 54.

34. STEWART, W. H., HOFFMAN, W. J., and GHISELIN, F. H. Cinefluorography. AM. J. ROENTGENAL. & RAD. THERAPY, 1937, *38*, 465–469.

35. STEWART, W. H., and GHISELIN, F. H. Paradoxical movement of diaphragm and mediastinal shift; cinefluorographic studies. AM. J. ROENTGENOL. & RAD. THERAPY, 1939, *41*, 926–930.

36. STEWART, W. H., BREIMER, C. W., and MAIER, H. C. Cineroentgenographic diagnosis of congenital and acquired heart disease. AM. J. ROENTGENOL. & RAD. THERAPY, 1941, *46*, 636–640.

37. SUSSMAN, M. L., STEINBERG, M. F., and GRISHMAN, A. Multiple exposure technique in contrast visualization of the cardiac chambers and great vessels. AM. J. ROENTGENOL. & RAD. THERAPY, 1941, *46*, 745–747.

38. TORELLI, G. Lo stato attuale della roentgencinematographia. *Ann. Ist. Carlo Forlanini*, 1939, *3*, 160–170.

Vol. 57, No. 1

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

Editor: Lawrence Reynolds, M.D. *Assistant Editor:* Ruth Bigelow, B.S.

Editorial Board: A. C. Christie, M.D. E. H. Skinner, M.D. Lauriston S. Taylor
Advisory Board for Pathology: Eugene L. Opie, M.D.
Collaborating Editors: The Officers and Committee Members of the Societies of which this Journal is the official organ, whose names appear on this page, are considered collaborating editors of this Journal. *Foreign Collaborators:* Gösta Forssell, M.D., Stockholm, R. Ledoux-Lebard, M.D., Paris.
Publisher: Charles C Thomas, 301–327 East Lawrence Avenue, Springfield, Illinois.

Issued Monthly. Subscription $10.00 per year, $11.00 in Canada and $12.00 in foreign countries. Advertising rates submitted on application: Editorial office, 110 Professional Building, Detroit, Mich., Office of publication, 301–327 East Lawrence Avenue, Springfield, Ill. Information of interest to all readers will be found on page iv.

Officers and Standing Committees

AMERICAN ROENTGEN RAY SOCIETY

President: Raymond C. Beeler, Indianapolis, Ind.; *President-Elect:* J. Bennett Edwards, Leonia, N. J.; *1st Vice-President:* W. Walter Wasson, Denver, Colo.; *2nd Vice-President:* Paul A. Bishop, Philadelphia, Pa.; *Secretary:* H. Dabney Kerr, University Hospital, Iowa City, Iowa; *Treasurer:* Wendell G. Scott, 510 South Kingshighway Blvd., St. Louis 10, Mo.

Executive Council: R. C. Beeler, J. B. Edwards, W. W. Wasson, P. A. Bishop, H. D. Kerr, W. G. Scott, Lawrence Reynolds, H. F. Hare, R. A. Arens, J. T. Case, L. C. Kinney, Ross Golden, B. P. Widmann, V. W. Archer, H. M. Weber, Chairman, Mayo Clinic, Rochester, Minn.

Program Committee: H. D. Kerr, Iowa City, Iowa, Lawrence Reynolds, Detroit, Mich., J. T. Case, Chicago, Ill., R. A. Arens, Chicago, Ill., H. M. Weber, Rochester, Minn., J. B. Edwards, Chairman, 144 Woodridge Place, Leonia, N. J.

Publication Committee: J. C. Bell, Louisville, Ky., R. J. Reeves, Durham, N. C., J. T. Case, Chairman, Chicago, Ill.

Finance Committee: W. W. Furey, Chicago, Ill., E. L. Jenkinson, H. F. Hare, Chairman, Boston, Mass.

Committee on Scientific Exhibits: Wilbur Bailey, Los Angeles, Calif., C. A. Good, Jr., Rochester, Minn., R. A. Arens, Chairman, Michael Reese Hospital, Chicago, Ill.

Representative on National Research Council: Barton R. Young, Philadelphia, Pa.

Editor: Lawrence Reynolds, 110 Professional Building, Detroit 1, Mich.

Assistant Editor: Ruth Bigelow, 110 Professional Building, Detroit 1, Mich.

Editorial Board: A. C. Christie, E. H. Skinner, Lauriston S. Taylor.

Advisory Board for Pathology: Eugene L. Opie.

Forty-eighth Annual Meeting: Haddon Hall, Atlantic City, N. J., Sept. 16–19, 1947.

AMERICAN RADIUM SOCIETY

President: Charles L. Martin, Dallas, Texas; *President-Elect:* A. N. Arneson, St. Louis, Mo.; *1st Vice-President:* Maurice Lenz, New York, N. Y.; *2nd Vice-President:* William S. MacComb, New York, N. Y.; *Secretary:* Hugh F. Hare, 605 Commonwealth Ave., Boston, Mass.; *Treasurer:* Leland R. Cowan, 606 Medical Arts Bldg., Salt Lake City, Utah.

Executive Committee: Frederick W. O'Brien, Chairman, Boston, Mass., Hayes Martin, New York, N. Y., William E. Costolow, Los Angeles, Calif.

Program Committee: A. N. Arneson, Chairman, St. Louis, Mo., William S. MacComb, New York, N. Y., Eugene T. Leddy, Rochester, Minn., Franklin B. Bogart, Chattanooga, Tenn., Orville N. Meland, Los Angeles, Calif.

Publication Committee: Edward H. Skinner, Chairman, Kansas City, Mo., Zoe A. Johnston, Pittsburgh, Pa., Wilbur Bailey, Los Angeles, Calif.

Research and Standardization Committee: Edith H. Quimby, Chairman, New York, N. Y., James L. Weatherwax, Philadelphia, Pa., Robert B. Taft, Charleston, S. C.

Education and Publicity Committee: Edwin C. Ernst, Chairman, St. Louis, Mo., Howard B. Hunt, Omaha, Neb., G. Allen Robinson, New York, N. Y.

Janeway Lecture Committee: Douglas Quick, Chairman, New York, N. Y., E. H. Skinner, Kansas City, Mo., G. Failla, New York, N. Y., L. A. Pomeroy, Cleveland, Ohio, F. W. O'Brien, Boston, Mass., F. E. Adair, New York, N. Y.

Representatives on American Board of Radiology: Douglas Quick, New York, N. Y., B. P. Widmann, Philadelphia, Pa., F. W. O'Brien, Boston, Mass.

Committee on Arrangements: To be appointed.

Twenty-ninth Annual Meeting: Seaside Hotel, Atlantic City, N. J., June 9–10, 1947.

EDITORIALS

BRONCHIECTASIS FOLLOWING PRIMARY TUBERCULOSIS

BRONCHIECTASIS is not an uncommon finding and the various infections of childhood have long been suggested as an etiological factor in bringing about this condition. It was not until within recent years that some communications appeared in the literature indicating that certain pathologic factors seen during the course of primary tuberculosis might conceivably produce ectasis.

Jones and Cournand,[1] as early as 1933, discussed the shrunken lobe associated with bronchiectasis. These investigators described the pathogenic factors involved, mentioning among others enlarged lymph nodes in primary tuberculosis as being a possible cause, particularly in involvements of the middle lobe.

Richards,[2] at the Thirty-third Annual Meeting of the American Roentgen Ray Society in 1932, called attention to the interpretation of the triangular basal shadows in roentgenograms of the chest and their association with bronchiectasis. Jones, Rafferty and Willis,[3] in 1942, mentioned the fact that primary tuberculosis is often complicated by bronchial tuberculosis with atelectasis. Their study revealed that the roentgenograms of 12 per cent of 716 children admitted to the Maybury Sanatorium over a five year period showed a dense, more or less homogeneous and usually segmented or lobar shadow that suggested obstructive pneumonitis. Forty-two of the patients with this syndrome were examined bronchoscopically and a large percentage, namely 31 per cent, of them were found to have tuberculous involvement of a bronchus which was present as ulceration, granulation tissue or tuberculoma, or as extrinsic pressure from enlarged tuberculous lymph nodes. The presence of these lesions appeared to interfere with the normal bronchial cleansing, and in the analysis of these cases it was observed by examination of the bronchograms that certain of these children had bronchiectasis in the region of the obstructive pneumonitis.

Recently,[4] a few years after this demonstration, a number of these children were recalled for further study, together with other children having the same phenomenon but more recently admitted. These children were re-examined for an evaluation of their present condition and especially to make a survey of their bronchial system by bronchography.

In this study 37 children were examined. These children varied in ages from two to twelve and the average age was five and one-half years. In this group the duration of the obstructive lesions could be followed from diminution to clearing and usually the time of onset could be placed rather accurately by the symptoms. The duration of the obstructive lesions appeared to have some relation to the establishment of the bronchiectasis.

In an analysis of these cases the time

[1] Jones, O. R., and Cournand, A. The shrunken pulmonary lobe with chronic bronchiectasis. *Am. Rev. Tuberc.*, 1933, *28*, 293–316.

[2] Richards, G. E. Interpretation of the triangular basal shadows in roentgenograms of the chest. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *30*, 289–295.

[3] Jones, Edna M., Rafferty, T. N., and Willis, H. S. Primary tuberculosis, complicated by bronchial tuberculosis with atelectasis (epituberculosis). *Am. Rev. Tuberc.*, 1942, *46*, 392–406.

[4] Jones, Edna M., Peck, W. M., and Willis, H. S. Bronchiectasis following primary tuberculosis. *Am. J. Dis. Child.*, 1946, *72*, 296–309.

relationship showed that by far the larger number of children in whom bronchiectasis developed had had a pulmonary lesion longer than one year.

The lesion was distributed equally between the two lungs, 12 children showing ectasis on the right and 12 on the left. There appeared to be a preference for a localization in the anterolateral branch of the upper lobe bronchus and in the apical branch of the lower lobe bronchus. In this group of cases no ectasis was discovered in the lingula. This finding was of considerable interest "because bronchiectasis in tuberculous adults has been found quite commonly in this part of the lung."

The roentgenographic pattern in these cases was often quite complex, depending on the fluctuation of the associated bronchial disease. "In those cases in which the lesions were viewed in the inception they characteristically showed the parenchymal infiltration and the enlarged lymph nodes of the primary complex, but with the appearance of bronchial obstruction a homogeneous opacity, notable for its strict segmental arrangement, developed tributary to the bronchial lesion and usually in the neighborhood of the primary focus." Often clearing of these areas of true atelectasis occurred without residual damage; but apparently the duration and the degree of obstruction and the extent of the original primary disease and, above all, of the amount of damage therefrom, appeared to be the factors vital in influencing the residual bronchial damage.

The symptoms in these cases were variable. In one group the most common and consistent symptom was wheezing; cough, expectoration, bouts of fever and at times acute illness with high fever also presented themselves. However, these symptoms disappeared with the clearing of the pneumonitis and the disappearance of the tuberculous process.

In some cases the bronchial dilatation was essentially without symptoms or the indications of classic bronchiectasis. The fact that manifest bronchial dilatation existed in 24 of the children examined for a considerable time with relatively few symptoms of bronchiectasis was somewhat bewildering, and Willis and his coworkers raised the question as to whether silent bronchiectasis is common, whether years may be required before symptoms develop, and whether bronchiectasis of this segmental type and of the origin which they were investigating commonly causes symptoms or is actually of much clinical importance.

They arrived at the conclusion that bronchiectasis occurs in an unusually large number of children having pulmonary tuberculosis. The question might be raised as to whether an absolute proof has been presented in the analysis of their cases to show that bronchial obstruction from tuberculosis predisposes to or causes the ectasis. However, it is quite doubtful that in their group of children a nonclinical bronchiectasis prior to the tuberculous infection existed when it was demonstrated that in every instance the segment of the lobe having the bronchiectasis was the previous site of the tuberculous process. From an analysis of all of their data they came to the conclusion that pulmonary tuberculosis in children is frequently complicated by tuberculosis of a bronchus, that these bronchial lesions are often associated with obstructive pneumonitis and that a large percentage of children who have shown obstructive lesions associated with tuberculosis of a bronchus revealed bronchiectasis in the segment or lobe involved in the obstruction, and moreover the dilatation of the bronchus extends from the root of the lung to the periphery. From an analysis of their study they arrived at the conclusion that pulmonary tuberculosis in children appears to be a common cause of bronchiectasis.

At the recent meeting of the American Roentgen Ray Society in Cincinnati Dr. Eduardo Rivero of Havana presented in the Scientific Exhibit a series of bronchograms of children with primary tuberculosis

showing the alterations of the bronchi caused by infiltrations of the bronchus by the tuberculous process and also compression of the bronchus from enlarged lymph nodes.

Therefore, from these studies by various observers one can only come to the conclusion that pulmonary tuberculosis is a not infrequent cause of bronchiectasis observed in childhood.

ADOLF LIECHTI
1898–1946

WORD has been received from Europe, that one of the most outstanding Swiss radiologists, Dr. Adolf Liechti, professor of radiology of the University of Bern, died on August 28, 1946, in Bern, when only forty-eight years old. His life and career came to a sudden end, due to heart failure, after he had suffered for more than twelve years from a slow progressive spondylarthrosis ankylopoietica. He is survived by his wife Martha Liechti-Lorétan and a son and daughter.

Professor Liechti was born April 18, 1898 in Burgdorf, Switzerland, the son of a Swiss farmer. He attended the gymnasium in Burgdorf and then enrolled in the Med-

ical School of the University of Bern, from which he graduated in 1924. After his internship at the Inselspital, he took up postgraduate work in dermatology and internal medicine under Professor Naegeli and Professor von Sahli.

His interest was not confined to medicine, but extended to physics, electrical engineering and radiology. His first studies of roentgen rays were made under Dr. Steiger in Bern and culminated in his thesis, "Zur Beeinflussung von bioelektrischen Potentialdifferenzen durch Röntgenstrahlen."

Because Switzerland at that time did not afford the opportunity for a thorough study of radiology he went in 1927 to Hamburg to work with Professor Holthusen, who was then director of the Roentgen Institute of the University. Here he published in joint authorship with Dr. H. Jacobi several papers dealing with basic qualitative and quantitative measurements of roentgen rays and the effect of radiation upon various biological subjects.

He returned to Bern in 1929, became first head of the Roentgen Institute of the University, and then professor of radiology. Here he designed and built one of the most efficient diagnostic departments in all Central Europe, including a huge research laboratory which was his special pride. In addition to his obligations as teacher, as director of the Inselspital's X-ray department, as radiologist of the Salemspital, Zieglerspital and of his own private roentgen institute, he found time to carry on a very intensive research program. More than fifty printed articles dealing with physical, biological and various diagnostic problems and with evaluation of deep and contact therapy carry his name. He is the author of the most outstanding textbook on roentgen physics in the German language and of a monograph on diseases of the spine. He also designed many technical devices, such as a "Funkenzähler" for the detection of very small quantity radiation and an apparatus for taking roentgenograms of the heart in systole or diastole.

Professor Liechti was past president of the Swiss Roentgen Society, a member of the Swiss Medical Society and of many other local medical organizations. He was co-editor of the Swiss "Radiologia clinica," of the Swedish "Acta radiologica," of the German "Strahlentherapie," and other journals. He was a major of the medical corps of the Swiss army. Many of his former students and associates have become radiologists, because of the interest he inspired in them for this branch of medicine.

With the same dogged determination with which he fought his incurable disease, he fought for his political and social convictions. Surrounded by Nazi Germany, he had the courage to fight on the side of the democratic scientists of Europe and helped many foreign students and colleagues who had to leave their native country because of religious, racial and political persecution. As early as 1935 he joined the central committee of "Frena," a Swiss organization to fight Nazi activities in Switzerland.

Professor Liechti's death is not only a great loss to his own country and to Europe, but to the medical world at large. His memory will long be cherished by his students, colleagues, patients and friends alike.

EUGENE F. LUTTERBECK

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Haddon Hall, Atlantic City, N. J., Sept. 16–19, 1947.

American College of Radiology
Secretary, Mac F. Cahal, 20 N. Wacker Drive, Chicago 6. Annual meeting: 1947, to be announced.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio. Annual meeting: 1947, to be announced.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: Seaside Hotel, Atlantic City, N. J., June 9–10, 1947.

Alabama Radiological Society
Secretary, Dr. John D. Peake, Mobile Infirmary, Mobile Ala. Next meeting time and place of Alabama State Medical Association.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1946.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Section on Radiology, California Medical Association
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Robert M. Lowman, Grace-New Haven Community Hospital, New Haven 11, Conn. Meets bimonthly on second Thursday, at place selected by Secretary.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

Radiological Section, Los Angeles Co. Med. Assn.
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. H. Levy, 1354 Carroll St., Brooklyn 13, N. Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Chicago Roentgen Society
Secretary, Dr. T. J. Wachowski, 310 Ellis Ave., Wheaton, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. Carroll C. Dundon, 11311 Shaker Blvd., Cleveland, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Denver Radiological Club
Secretary, Dr. W. C. Huyler, 1619 Milwaukee, Denver 6, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida Radiological Society
Secretary, Dr. J. M. Dell, Jr., 333 W. Main St., S., Gainesville, Fla. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. J. A. Campbell, Indiana University Hospitals, Indianapolis 7. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association, the other in the fall.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Neb. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

* Secretaries of societies not here listed are requested to send the necessary information to the Editor.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. W. H. Seward, Orange Memorial Hospital, Orange, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. Hemphill, 323 Professional Bldg., Charlotte 2, N. C. Meets in May and October.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

CENTRAL NEW YORK ROENTGEN RA SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. P. E. Russo, 230 Osler Bldg., Oklahoma City, Okla. Three regular meetings annually.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. C. L. Stewart, Jefferson Hospital. Meets first Thursday of each month, October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. L. M. J. Freedman, 115 South Highland Ave. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

PORTLAND ROENTGEN CLUB
Secretary, Dr. Selma Hyman, University of Oregon Medical School, Portland, Oregon. Meets monthly 2d Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September.

SAN DIEGO ROENTGEN SOCIETY
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph Levitin, 516 Sutter St., San Francisco 2, Calif. Meets monthly on the third Thursday at 7:45 P.M., first six months of the year at Lane Hall, Stanford University Hospital, and second six months at Toland Hall, University of California Hospital.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

UTAH RADIOLOGICAL CONFERENCE
Secretary, Dr. Henry H. Lerner, School of Medicine, University of Utah, Salt Lake City 1. Meets 1st and 3rd Thursdays monthly from 7:30 to 10 P.M., Salt Lake County General Hospital, September to June.

UTAH STATE RADIOLOGICAL SOCIETY
Secretary, Dr. M. Lowry Allen, Judge Bldg., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-RAY STUDY CLUB OF SAN FRANCISCO
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly, third Thursday evening.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

MEXICO

SOCIEDAD MEXICANA DE RADIOLOGIA Y FISIOTERAPIA
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

BRITISH EMPIRE

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE ROENTGEN SOCIETY
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

FACULTY OF RADIOLOGISTS
Secretary, Dr. M. H. Jupe, 23 Welbeck St., London, W.1 England.

SECTION OF RADIOLOGY AND MEDICAL ELECTRICITY, AUSTRALASIAN MEDICAL CONGRESS
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

RADIOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Keith Hallam, St. George's Hospital,

K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 2100 Marlowe Ave., Montreal 28, Que. Meetings January and June.

Société Canadienne-Francaise d'Électrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306 Lima, Peru. Meetings held monthly except during January, February and March, at the Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Continental Europe

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary: MUDr. Roman Blána, Praha XII, Korunní 160, Czechoslovakia.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. Babaiantz, Geneva.
Secretary for German language, Dr. Max Hopf, Effingerstrasse 49, Bern. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

MEDICAL AND SURGICAL SYMPOSIUM

The Fourth Annual Medical and Surgical Symposium sponsored by the Watts Hospital Staff of Watts Hospital, Durham, North Carolina, will be held at the Hospital on Wednesday and Thursday, February 12 and 13, 1947. The following program will be presented:

Wednesday, February 12, 1947

11:00 A.M. Clinico-pathological Conference, presented by Sidney Farber, M.D., Department of Pathology, Harvard Medical School, Boston, Mass., and Charles D. May, M.D., Associate in Pediatrics, Harvard Medical School, Boston, Mass.

2:30 P.M. Surgical Aspects of Diabetes, Leland S. McKittrick, M.D., Boston, Mass.

3:30 P.M. Recent Advances in the Study and Treatment of Diabetes, Howard F. Root, M.D., Boston, Mass.

8:00 P.M. Viral Hepatitis, Joseph Stokes, Jr., M.D., Professor of Pediatrics, University of Pennsylvania Medical School, Philadelphia, Pa.

9:00 P.M. Pericardial Scars, Francis C. Wood, M.D., Assistant Professor of Medicine, University of Pennsylvania Medical School, Philadelphia, Pa.

Thursday, February 13, 1947

11:00 A.M. Clinico-pathological conference, presented by Colonel J. E. Ash, Medical Corps, Director, Army Institute of Pathology, Washington D. C., and Wallace Yater, M.D., Former Professor of Medicine at Georgetown University.

2:30 P.M. Some Clinical and Physiologic Aspects of Portal Cirrhosis, Albert M. Snell, M.D., Mayo Clinic, Rochester, Minn.

3:30 P.M. The Current Status of Calcium Penicillin in Beeswax and Peanut Oil, Monroe J. Romansky, M.D., George Washington University School of Medicine, Washington, D. C.

8:00 P.M. The Present Status of Effective Specific Therapy Based on Exact Hematological Diagnosis, Charles A. Doan, M.D., Dean, College of Medicine, Ohio State University, Columbus, Ohio

UROLOGY AWARD

The American Urological Association offers an annual award "not to exceed

$500" for an essay (or essays) on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to residents in urology in recognized hospitals.

For full particulars write the Secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis, Tennessee. Essays must be in his hands before May 1, 1947.

The selected essay (or essays) will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Hotel Statler, Buffalo, New York, June 30–July 3, 1947.

Committee on Scientific Research
Miley B. Wesson, Chairman
Judson B. Gilbert
Anson L. Clark

AMERICAN RADIUM SOCIETY

The Twenty-ninth Annual Meeting of the American Radium Society will be held at the Seaside Hotel in Atlantic City on June 9 and 10, 1947.

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Genitourinary System

Higgins, C. C., and Rambousek, E. S.: Diverticula of urethra in women 126
Kickham, C. J. E.: Obstruction of ureter in children 126
Woodruff, S. R., and Begner, J. A.: Sacrococcygeal teratoma in an infant of five months causing acute urinary obstruction. 126
Pentecost, C. L., and Pizzolato, P.: Involvement of genito-urinary tract in leukemia .. 127
Wilhelm, S. F.: Rectourinary fistula 127

Nervous System

Mayfield, F. H., and Devine, J. W.: Causalgia 128

Skeletal System

Paggi, L. C., and Hull, E.: Metastatic granuloma venereum 128
Woolley, P. V., Jr., and McCammon, R. W.: Bone growth in congenital myxedema 129
Scott, W.: March fractures 129
Hamilton, J. F.: Osteoid osteoma 130
Saypol, G. M., and Slattery, L. R.: Observations on displaced fractures of the hand .. 131
Cramer, F., and McGowan, F. J.: Role of nucleus pulposus in pathogenesis of so called "recoil" injuries of spinal cord 131
Cohn, B. N. E.: Total and partial patellectomy 132
Schmier, A. A.: Excision of fractured patella. 132
Cave, E. F., Rowe, C. R., and Yee, L. B. K. Chondromalacia of patella 133
Nachlas, I. W., and Olpp, J. L.: Paraarticular calcification (Pellegrini-Stieda) in affections of knee 134
Ver Brugghen, A.: Massive extrusions of lumbar intervertebral discs 135

Blood and Lymph System

Clarke, H. M.: Primary carcinoma of extremity 136
Voris, H.: Subdural hematoma 136
Lesh, R. E.: Mesenteric thrombosis complicated by dermoid cyst 137

General

Wilson, D. A.: Tumors of subcutaneous tissue and fascia 138

ROENTGEN AND RADIUM THERAPY

Snitman, M. F.: Individualization in management of carcinoma of maxillary sinus 138
Smith, G. V., and Dresser, R.: Carcinoma of uterine cervix 139
McLennan, C. E.: Results of various types of treatment in adenocarcinoma of the endometrium 140
Parks, T. J.: Carcinoma of ovary treated preoperatively with deep x-ray 140
Bethell, F. H.: Leukemia 141
Uhlmann, E. M.: Alpha rays in treatment of wounds 141
Prudente, A.: Postoperative prophylaxis of recurrent mammary cancer with testosterone propionate 142
Treves, N., Abels, J. C., Woodard, Helen Q., and Farrow, J. H.: Effects of orchiectomy on primary and metastatic carcinoma of breast 143
Jones, H. W., Jr., and Neill, W., Jr.: Treatment of carcinoma of cervix during pregnancy 143

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

GENITOURINARY SYSTEM

HIGGINS, CHARLES C., and RAMBOUSEK, EDWARD S. Diverticula of the urethra in women; review of 12 cases. *J. Urol.*, May, 1945, *53*, 732–739.

The diagnosis of diverticulum of the female urethra is being made more frequently. Congenital diverticula are rare. Most diverticula of the female urethra are acquired following infections of the urethral glands with sealing off of their ducts, abscess formation and rupture into the urethra. They may also follow strictures, stones or urethral instrumentation. The common symptoms are dysuria, discharge of pus from the urethra followed by symptomatic relief and a palpable mass. The diagnosis is made by palpation of the urethra, urethroscopy and urethrography.

The authors report a series of 12 cases with urethrograms illustrating 7 of the 12 cases. In all cases the symptoms were of fairly long duration and all the patients but 2 had a history of recurring attacks of cystitis. All complained of a painful urethral mass and all but 2 complained of a purulent urethral discharge. Surgical excision is the treatment of choice.—*R. M. Harvey.*

KICKHAM, C. J. E. Obstruction of the ureter in children. *J. Urol.*, June, 1945, *53*, 776–780.

Many of the kidney diseases of adult life which lead to sacrifice of the kidneys have their origin in congenital conditions which could have been discovered and corrected in early life. This is particularly true of congenital ureteral blockage with its subsequent obstructive effects. In the past some justification for failure to recognize these congenital lesions was existent due to lack of precision instruments and hesitancy on the part of the physician to subject a young patient to urethral instrumentation. The present advanced development of the armamentarium of the urologist now precludes such justification. The study of urological conditions in the young has been greatly facilitated by the use of intravenous urography.

Symptoms suggesting possible serious urinary tract disease in the young are persistent pus in the urine, enuresis, hematuria, enlargement of the kidney and abdominal pain of urinary tract origin. Campbell has reported congenital obstructions of the ureter in 1 per cent of postmortem examinations of the urinary tract in children. This leads to infection and the poor drainage to hydronephrotic destruction of the kidney. The most frequent sites of obstruction are at the ureterovesical and ureteropelvic junctions. Many of these obstructive lesions are amenable to surgery. This must be done before irreparable renal damage has occurred. Successful results of the surgery must be checked by urography. The author discusses various operative procedures for the correction of the common obstructive lesions, strictures, aberrant vessels and high insertion of the ureters.—*R. M. Harvey.*

WOODRUFF, S. R., and BEGNER, J. A. Sacrococcygeal teratoma in an infant of five months causing acute urinary obstruction. *J. Urol.*, Aug., 1945, *54*, 177–181.

Congenital deformities are prone to occur at the caudal end of the fetus. Sacrococcygeal teratomata are approximately four times as common in female infants. Seventy-two cases have been reported since 1924 and 65 of these had an obvious deformity on external examination at birth. The tumor lies between the rectum and the vertebral column. It tends to grow outward although it may rarely grow into the pelvic cavity and can only be detected by rectal examination. The authors present a case of the latter or internal type.

Their patient was a five months' old male infant who was admitted to the hospital with a history of having been unable to void for three weeks. The onset had been acute and the child had required daily regular catheterization since the onset of symptoms. By rectal examination a smooth mass could be palpated pushing the rectum anteriorly and almost completely filling the true pelvis. An intravenous urogram showed normal pelves and calices of both kidneys. The right ureter was displaced laterally. A cystogram showed displacement of the bladder to the

right and in a cephalad direction. On the lateral view the bladder was cut off at the symphysis. A barium enema showed an extrinsic pressure effect on the rectosigmoid.

Operation disclosed a pear-shaped tumor firmly attached to the anterior surface of the lower sacrum and coccygeal segments. The upper portion of the tumor mass was cystic. The tumor was removed but the child died less than twenty-four hours after operation. Histopathological examination revealed pancreatic tissue, lung tissue, ciliated epithelium, smooth muscle, renal and brain tissue.

This tumor should always be considered in the differential diagnosis of acute urinary retention in the infant and a rectal examination should not be omitted.—*R. M. Harvey.*

Pentecost, C. L., and Pizzolato, P. Involvement of the genito-urinary tract in leukemia, with report of a case of involvement of the urinary bladder. *J. Urol.*, May, 1945, *53*, 725–731.

According to published reports the urinary tract is involved in about 50–60 per cent of cases of leukemia. Although symptoms referable to the urinary tract may be the presenting symptom such symptoms do not apparently occur in most of the cases in which there is leukemic involvement of the urinary tract. The urinary bladder is seldom invaded by leukemia.

The authors report a case of a nineteen year old male Negro who was admitted with chief complaints of straining after urination, and terminal pain and hematuria. Physical examination revealed anemia and weight loss with a palpable spleen. Urinalysis revealed microscopic blood. Cystoscopic examination showed such marked blood oozing that the field was almost obscured. Numerous finger-like projections of the bladder mucosa were seen in all areas and a tentative diagnosis of papillary carcinoma of the bladder was made. There was constant marked bladder irritability during the entire hospitalization. The patient developed high fever and left costo vertebral angle pain following the original cystoscopy and died before a second examination was performed.

Postmortem examination showed extensive leukemic involvement of the mesenteric lymph nodes, liver, spleen, gastrointestinal tract and kidneys. The bladder showed a dense infiltration of lymphocytes in the submucosa and mucosa. The final diagnosis was chronic lymphatic leukemia. The diagnosis was not made antemortem because through error no blood count was made. This case illustrates that group of cases of leukemia in which the presenting symptoms are all referable to the urinary tract. This was somewhat unusual in that the disease was so far advanced and so many systems were involved with the disease.—*R. M. Harvey.*

Wilhelm, Seymour F. Rectourinary fistula. *Surg., Gynec. & Obst.*, Oct., 1944, *79*, 427–433.

Improvement in diagnostic methods and prompt surgical intervention have materially lessened the incidence of inflammatory fistulas between the rectum and the urinary passages. The advent of perineal and transurethral-prostatic surgery, however, has more than compensated for this loss. This report deals with 18 cases of rectovesical and rectourethral fistula observed within the last twenty-one years at the Beth Israel and the Montefiore Hospitals.

Etiology. The causative factor in these 18 cases was determined as follows: traumatic, 8 cases; inflammatory, 4 cases; neoplastic, 6 cases; doubtful, 1 case.

Symptoms and Diagnosis. The passage of gas and fecal matter with the urine or the presence of urine in the rectum indicates the existence of a communication between the bowel and the urinary passages. Gas alone in the urine, however, is known to occur in certain rare bladder infections. Some fistulas possess a ball-valve-like action allowing free passage in but one direction.

The injection of colored or radiopaque solutions into the bladder or rectum is also of value in proving the existence of an abnormal communication. However, short fistulas of large diameter between the rectum and the urinary passages are themselves rarely visualized on the roentgenogram. A contrast solution injected into the bladder or urethra flows freely through such a wide communication, speedily outlining the rectum and sigmoid colon. On the other hand, the longer and more tortuous fistulas, due usually to inflammatory disease, may often be clearly delineated by the ingestion of barium or the injection of iodized oil.

Treatment. Acute fistula—The immediate treatment of a recent rectourinary fistula is concerned with: (1) control of hemorrhage, (2) urinary drainage by urethral or suprapubic catheter, (3) constipation by diet and drugs,

(4) intestinal bacteriostasis (sulfasuxidine, sulfaguanidine).

Results. Simple measures, such as an indwelling catheter and induced constipation, resulted in permanent closure in 3 of the 6 cases of acute fistula. On the other hand, a rectourethral fistula, 1 cm. in diameter, resulting from a transurethral prostatectomy, persisted despite colostomy and suprapubic cystostomy. This fistula, as well as the two which complicated perineal prostatectomy, was short and of large diameter, the posterior urethra and rectum being in close apposition. Such fistulas are less likely to close spontaneously than are the long narrow tortuous tracts usually resulting from inflammatory disease. Both fistulas which followed perineal prostatectomy failed to heal despite urethral and suprapubic catheter drainage but were later cured by perineal reparative operation.

Chronic fistula. The treatment of chronic rectourinary fistula may be considered under 3 headings: (1) diversion of the fecal stream, (2) diversion of the urinary stream, (3) surgical repair.—*Mary Frances Vastine.*

NERVOUS SYSTEM

MAYFIELD, FRANK H., and DEVINE, JOHN W. Causalgia. *Surg., Gynec. & Obst.*, June, 1945, *80*, 631–635.

The term causalgia was used in 1865 by Mitchell, Morehouse and Keen to describe a bizarre syndrome resulting from injuries to peripheral nerves. Its cardinal features are the subjective complaint of burning pain in association with trophic and vasomotor changes in the injured extremity. The pathology and pathogenesis of causalgia are unknown. Indeed, there is some confusion as to just what constitutes this syndrome. Some writers believe that causalgia, Sudeck's atrophy, painful osteoporosis, and other so-called " minor causalgias," and, indeed, painful "phantom limb," are different clinical manifestations of the same underlying disorder. The similarity, however, is remote and the authors are of the opinion that causalgia varies sufficiently in severity to be considered a clinical entity.

Summary and Conclusions

In a study of 15 cases of causalgia the following observations were made:

1. Burning pain is a constant complaint and is usually immediate in onset.

2. The median or sciatic nerve was involved in each instance and the lesion was incomplete.

3. Certain patients showed vasoconstriction in the causalgic limb; others showed vasodilatation.

4. The trophic changes and response to warm or cold moisture and to hyperthermia varied depending upon the blood flow.

5. Twelve patients were relieved by sympathectomy of the involved limb; 1 was cured by artificial fever therapy; and 2 recovered spontaneously.

6. Direct surgical attack (neurolysis) upon the injured nerve (which was performed on 5 cases) and periarterial sympathectomy at the level of the injury (performed in 3) have not been of benefit.

7. Procaine block of the sympathetic chain affords complete relief temporarily and should be repeated 2 or 3 times although no case in this series has had any lasting benefit from this procedure.

8. Sympathectomy (preganglionic) gave immediate and lasting relief to 12 patients.

9. The site of injury must be included in the sympathectomy.

10. Sympathetcomy should be done early to prevent stiffness of joints due to disuse.

11. The personality changes always present during the painful stages are secondary to the pain. There is no evidence in these cases that there is a predisposing constitutional psychic factor in causalgia.—*Mary Frances Vastine.*

SKELETAL SYSTEM

PAGGI, L. C., and HULL, EDGAR. Metastatic granuloma venereum, *Ann. Int. Med.*, April, 1944, *20*, 686–695.

A case of granuloma venereum with metastatic lesions of the left clvaicle, right scapula, right sixth rib and the left tenth rib is reported. The bone lesions as seen on the roentgenogram were osteolytic and a biopsy of one of them showed the characteristic pathological lesions of granuloma venereum. Donovan bodies were abundantly demonstrable in ulcerative lesions on the cervix, labium majus and groin. Subcutaneous abscesses, two of which were followed by deep ulcers, occurred over each of the sites of bone involvement. Constitutional symptoms, usually slight or absent in cases of granuloma venereum, were very severe. Marked improvement of the lesions and disappearance of the constitutional symptoms followed therapy with a trivalent antimony compound.—*James J. McCort.*

Woolley, P. V., Jr., and McCammon, R. W. Bone growth in congenital myxedema; use of serial roentgenograms of the os radius in diagnosis and regulation of therapy during infancy. *J. Pediat.*, Sept., 1945, *27*, 229–235.

The appearance time of ossification centers, although it provides a useful index to thyroid function in the infant, does not serve as a practical guide to regulation of therapy in cretins, since the changes occur too slowly. The authors feel that a more practical guide is to be found in the measurement of linear growth of the long bones and in the degree of calcification of the epiphyseal plates. An infant deprived of its normal thyroxine will show a definite reduction in the rate of linear growth of the long bones plus increased density of the zones of provisional calcification. The rate of linear growth can be compared with normal curves. Woolley and McCammon used as their standards a growth curve constructed from four hundred measurements on the radii of babies with no evidence of thyroid dysfunction. They studied the radii in 5 cretins and in several other cases showing growth disturbance from other causes. The roentgenograms of 2 of the cretins are reproduced to show the typical changes. In addition to the absence of ossification centers normally present, the roentgenograms showed increased density of the zones of provisional calcification and radii shorter than the normal. It was found that under specific therapy bone growth occurred at a rate greater than normal for the first few months and then slowed to parallel the mean curve. At this point increased thyroid administration did not result in increased bone growth.

The authors feel that the dense epiphyseal plate is as accurate an index of congenital myxedema as is the retardation of the appearance of the centers of ossification. Although they claim this dense plate as seen in myxedema can be readily differentiated from that seen in scurvy, syphilis, lead poisoning, etc., they do not reveal their magic formula. They feel that decrease in linear growth of the long bones, as measured on the films, is a more sensitive index of thyroid deficiency than are the clinical signs of myxedema.

Thyroid deficiency can be differentitaed from other states characterized by growth retardation by a therapeutic administration of thyroid followed by serial roentgenograms of the long bones. If no growth occurs following the administration of thyroid the growth disturbance does not have a thyroid basis.

This test is not applicable after the second year when there is considerable slowing of bone growth and other tests of thyroid deficiency become more easily applicable.—*R. M. Harvey.*

Scott, Walter. March fractures. *Surg., Gynec. & Obst.*, Nov., 1945, *81*, 525–529.

The author reports some observations made during the treatment of 58 cases of march fracture involving the second or third metatarsal bones in the period from September 1, 1944, to February 10, 1945. All the patients were males who developed their trouble during some phase of their recruit schedule at a large Naval training center.

Etiology. Forty-eight of the 58 cases were studied from the standpoint of basal metabolic rate. The average rate was minus 10.72 per cent. The average pulse rate of this group was 62, and 57 per cent of the men were either tall or fat.

Clinical Course. There may be no roentgenographic evidence of the fracture until the seventh or tenth day following its acquisition. Roentgenologically, these fractures will fall into one of two groups as far as prognosis is concerned: (1) The smaller group (10 per cent) shows the fracture line to run completely across the shaft of the bone. (2) The larger group (90 per cent) will either show an incomplete fracture line or simply a periosteal shadow.

Treatment. The author's routine entails the principle of bed rest for a period of about five days and the administration of desiccated thyroid, 1 grain three times a day for a total of nineteen days. An examination is conducted daily with particular respect to the soft tissue swelling and it has been noted that the swelling will disappear gradually in two to five days. However, the foot will appear swollen for many weeks, due to the presence of periosteal callus at the fracture site. It is important to differentiate between these two (inflammatory and reparative) types of swelling. This can be done by means of the "pinch test." By pinching the skin between the thumb and index finger on both the normal and affected foot, a thickening or turgidity in the subcutaneous tissue will be noted if inflammatory edema is present. In most cases this test will become negative at the end of the third, fourth, or fifth day. Bed rest is continued for two days after the pinch test is negative. The patient is then allowed up and returned to

full duty with a supply of thyroid tablets sufficient to last a total of nineteen days. No other treatment is given.

Results. Mild, nondisabling symptoms were a complaint in half of the patients thus treated (these symptoms lasted from one to fourteen days).

Those who received thyroid showed better intermediate results than those who were treated by bed rest alone.

The final results were excellent, except for that group having complete fractures.

Conclusions. It is probable that a mild hypothyroid state may be a predisposing systemic cause in patients who develop march fractures. Cast treatment is not necessary, nor is it indicated except perhaps in those cases in which the fracture line is complete. Bed rest continued through the period in which the inflammatory swelling is disappearing and continued for two days thereafter is the treatment of choice. The administration of thyroid substance for a short period is apparently helpful.—*Mary Frances Vastine.*

HAMILTON, J. F. Osteoid osteoma, with case reports. *Surg., Gynec. & Obst.*, Nov., 1945, *81*, 465–474.

Osteoid-osteoma is a benign bone lesion, so named by Jaffe in 1935. He believes it to be a definite entity, and defined it as follows: "The lesion is a benign osteogenic tumor of slow growth. The original phase of its evolution in a given site seems to be the proliferation of the local bone-forming mesenchyme and particularly of its osteoblasts. Indeed, at an early stage the tumor may consist largely of a vascular mesenchymal substratum, closely packed with osteoblasts, although showing also a scattering of osteoclasts."

Including the author's 5 cases, a total of 58 patients with osteoid-osteoma have been studied.

Etiology. The etiology is unknown. Whether trauma is a factor in precipitating the condition has not been confirmed.

Clinical Data.

Age. The lesion is most commonly seen in the second and third decades.

Sex. The lesion is seen more frequently in males (approximately 3:2).

Symptomatology. Pain is the dominant presenting complaint in all patients. It is usually localized to a small area directly over the involved site. The pain and tenderness may antedate any other clinical or even roentgenographic evidence of the disease by several weeks or months.

Physical Findings. Exquisite "finger point" tenderness over a small area, rarely more than 1 cm. in diameter, is an almost constant finding. Swelling of the adjacent overlying soft tissue is present occasionally. This is due to congestion and edema of the adjacent periosteum. Local fever is very rare and redness of the skin is seldom noted. Occasionally, the patient will complain of a "knot" at the site of the lesion, which is due to a deposit of sclerotic reactive new bone beneath the periosteum.

Skeletal Sites. Osteoid-osteomas have been found in the following bones: tibia, fibula, femur, vertebra, humerus, ulna, skull, facial bone, phalanges, patella, calcaneus, talus, tarsal navicular, and ilium.

Roentgenographic Findings. There may be none even though symptoms have been present for months. A typical picture reveals an oval to round area from a few millimeters to 1 or 2 cm. in its greatest diameter, the center of which presents small area of rarefaction and condensation. Surrounding this is a narrow zone of condensation and, in turn, just outside of this is a zone of rarefaction. The surrounding parent bone is, as a rule, sclerosed for a variable depth. Osteoid-osteoma may occur in the cortex just beneath the periosteum, intracortically, or in spongy bone.

Pathology. The gross appearance of a typical osteoid-osteoma usually consists of blood-stained, gritty, friable, cancellus-like bone. Microscopically, there is much vascular, richly cellular, embryonal osteogenetic connective tissue.

Diagnosis. The important features are: (1) Pain of a dull, aching character, most annoying at night. (2) Pain localized, for the most part, to a small area, but which may be referred. (3) Tenderness directly over the site of the lesion, and usually confined to a small area 1 or 2 cm. in diameter. (4) Soft tissue swelling may or may not be present. (5) Bony tumefaction may be present. (6) Local fever and redness are rare. (7) No noteworthy systemic symptoms. (8) All routine laboratory tests are negative. (9) The majority of the patients are in the second or third decades of life.

Differential Diagnosis. (1) Sclerosing nonsuppurative osteomyelitis of Garré; (2) Brodie's intracortical bone abscess; (3) osteogenic sarcoma; (4) Ewing's endothelial myeloma.

Prognosis. The prognosis is uniformly good provided all of the lesion is excised.

Treatment. Surgical excision of the pathologic lesion is the treatment of choice provided the area is accessible to surgery.—*Mary Frances Vastine.*

Saypol, George M., and Slattery, Louis R. Observations on displaced fractures of the hand. *Surg., Gynec. & Obst.*, Nov., 1944, *79*, 522–525.

"*Baseball Finger.*" Simple hyperextension of the distal phalanx as obtained by means of a padded tongue depressor is unphysiological and will yield poor results. Immobilization of the involved finger with molded plaster or a well fitting aluminum splint, with flexion at the proximal interphalangeal joint and hyperextension at the distal interphalangeal joint has proved satisfactory in the authors' hands. The position is emphasized as it is believed that adequate hyperextension can be obtained only by relaxing the flexor tendon. Plaster should be snugly applied, and a dorsal section corresponding to the shape of the nail should be cut out so that the finger tip may be observed for circulation. Care in the original splinting with observation for the first twelve to twenty-four hours may make the difference between union of an avulsed fragment to its bed and a permanently "dropped finger." For cases seen after the first week, open operation is usually necessary.

Philanges. In the treatment of fractured phalanges, the authors have employed a method of fixed traction. A traction splint is incorporated in plaster. The spint is made of aluminum alloy and extends from the palm to just beyond the tip of the finger. The palmar portion has four flanges to facilitate incorporation in plaster. The shaft is the width of the finger and in its distal portion has two flanges forming a trough. In these flanges, parallel vertical notches 4 mm. apart engage the needle used for transfixion.

Metacarpals. Following closed reduction of the fracture by traction on the flexed finger, a Kirschner wire is passed through the heads of the affected and its adjacent metacarpal or metacarpals. In the case of fracture of the 5th metacarpal or the much rarer 2nd metacarpal, it is necessary to incorporate the emergent wire under tension in a plaster gauntlet, leaving all the metacarpophalangeal joints free. The latter serves as a means of external fixation, while the adjacent metacarpal serves as a fulcrum. In fractures of the 3rd and 4th metacarpals one may dispense with plaster, except to protect the protruding wire end from trauma. The method has the advantage of allowing immediate motion in all the joints of the affected and normal fingers. Other forms of traction through the phalanges result in prolonged disability due to the long period of immobilization. Using the latter methods, joint complications in the finger used for traction are sometimes more disabling than the fractured metacarpal itself.

Fractures Involving Joints. Fractures and fracture-dislocations involving joints in which more than 50 per cent of one surface is involved, frequently terminate in ankylosis. This complication, particularly in the proximal interphalangeal joint is so disabling that the patient may request amputation. Traction in the position of function, followed by active motion in three weeks will circumvent these sequelae in many instances. Traction reduces the displaced fragments, realines the joint surfaces, and at the same time maintains the joint spacing so that the tendency to heal across and ankylose is minimized.—*Mary Frances Vastine.*

Cramer, Fritz, and McGowan Francis J. The role of the nucleus pulposus in the pathogenesis of so called "recoil" injuries of the spinal cord. *Surg., Gynec. & Obst.*, Nov., 1944, *79*, 516–521.

In the condition known as "recoil" injury, the spinal cord may be severely damaged without demonstrable or commensurate injury to the spinal column. Diving accidents, or certain automobile accidents in which the head is whiplashed forward, producing a forceful anterior flexion of the cervical spine, are the usual causes. Less frequently, extreme retroflexion may cause similar lesions. This is commonly stated and believed to be due to the sudden massive dislocation of a vertebral body, which then recoils by reflex contraction of the antagonist muscles. This explanation has been employed for a number of years and is rather generally repeated in the books and literature on the subject but it appears to have been arrived at on purely theoretical grounds and on negative observations.

On the basis of the authors' findings, they believe that it is not the rather incredible excursion of a vertebral body virtually across the width of the spinal canal and back again, but rather it is the violent protrusion of the intervertebral disc by means of the hydraulic ram-like action of the nucleus pulposus or the rup-

ture of the latter when subjected to sudden intense compressive force, which causes the damage to the spinal cord.

Pancoast, Pendergrass and Shaeffer write, "In the cervical region, the severity of the trauma to the spine bears little relation to the cord injury. Often, there is comparatively little roentgenographic evidence of traumatism to the spine with the most severe cord injuries. On the other hand, there may be a serious fracture, with permanent dislocation, and marked displacement, and little or no cord disturbance. It is evident, therefore, that the seriousness of the injury depends entirely on the damage, present and remote, to the cord and nerve tissues."

The tendency toward compression by the vertebral column is countered by the nucleus pulposus. The latter is quasi-liquid and is tightly surrounded by the flexible but inelastic matrix of fibrocartilage constituting the disc. This inelastic capsule, consisting of the fibers of the annulus fibrosus and of the anterior and posterior longitudinal and the lateral ligaments, tends to maintain the over-all dimensions of the disc unaltered under normal conditions of stress and movement.

Summary and Conclusions

1. The report of a case of injury by diving into shallow water is presented. The cervical cord of the patient was almost completely severed anteriorly without any evidence of dislocation present of the spinal column.

2. At postmortem examination there was revealed a protruded intervertebral disc directly opposite the lesion present in the spinal cord.

3. The previously held theory that such lesions were due to a momentary extreme vertebral dislocation with immediate spontaneous reduction is considered improbable on anatomic grounds.

4. Violent protrusion of the intervertebral disc, due to compression of the nucleus pulposus, or herniation of the latter, is proposed as the true mechanism producing the injuries to the spinal cord in this special type of traumatic paralysis.—*Mary Frances Vastine.*

COHN, BERNARD N. E. Total and partial patellectomy; experimental study. *Surg., Gynec. & Obst.*, Nov., 1944, *79*, 526–536.

Total excision of the patella is not a new procedure. It was used as early as 1860 by Putz and by Fowler in 1871 for the treatment of habitual dislocation of the patella. Since 1837 many publications have appeared appertaining to the removal of the patella for fracture and for arthritis. Warnings against the adoption of such a procedure routinely, however, are beginning to appear in the literature. Bruce and Walmsley performed experimental excision of the patella in rabbits, and reported degenerative changes in the articular cartilage, especially in the patellofemoral surface.

The present work was begun in January, 1942, before the appearance of Bruce and Walmsley's paper. The purpose was to ascertain the effects, if any, of total and partial excision of the patella on the structure of the knee joint both in young and adult rabbits. Since it was believed that function could not be satisfactorily tested clinically in the rabbit, specimens of the quadriceps muscle were taken from animals of each group in order to study histologically any changes which might have been caused by malfunction.

Summary

1. Total patellectomy in the rabbit resulted in degenerative arthritis (osteoarthritis) of the tibiofemoral articulation. The pathologic changes were more marked in the adult than in the immature animal.

2. Partial patellectomy in the adult rabbit evoked early degenerative changes of the femoral condyles as manifested by a fibrous outer stratum.

3. Partial patellectomy in the young animal did not produce articular changes (at least during the twelve-month period of the experiments).

4. No clinical aberrations of function were noted in either the total or partial patellectomy series. Histologic study of the quadriceps muscle did not reveal any atrophic changes.

Conclusion. At the present state of our knowledge it would appear that total patellectomy in the human should not be performed except in those instances in which such a procedure cannot be avoided.—*Mary Frances Vastine.*

SCHMIER, ADOLPH A. Excision of the fractured patella. *Surg., Gynec. & Obst.*, Oct., 1945, *81*, 370–378.

In direct injuries, such as a fall on the flexed knee, the patella is particularly prone to fracture. In indirect injuries, such as a sudden violent contraction of the quadriceps, one of four

types of defect may occur to cause a disruption of the extensor mechanism of the knee. The patella may fracture; the quadriceps or patellar tendons may rupture; or the tibial tubercle may become avulsed.

The author's particular consideration is with the fractured patella. Therapy in the other three instances is clear cut. Avulsed tibial tubercles are readily reduced and may be fixed in position by a screw. Ruptures of the quadriceps or patellar tendons require open operation for repair. The end-results in all three of these instances are usually good without disabling sequelae.

Regarding treatment of the fractured patella, the author concludes:

1. The treatment of fractured patellae in the past by so many methods is indicative of dissatisfaction with all of them.

2. When one considers the possibility of fibrous union or nonunion, union with restricted mobility and instability, and union with secondary arthritic changes, one notes the shortcomings of open as well as closed reduction of the patella.

3. The high incidence of refracture of the patella, even following open reduction, reveals the need for a more effective method of treatment.

4. Excision of the patella, either partial or complete, is the method of choice in fractures.

5. Partial excision with removal of the small comminuted fragments alone is indicated only when a large uninvolved portion of the patella can be left in situ.

6. In all other instances of fracture, total excision is preferable for the following reasons: (*a*) rapid progressive uneventful recovery usually ensues; (*b*) postoperative immobilization and convalescence are shortened; (*c*) strong, stable, and freely movable knee joints are generally obtained; (*d*) even when complicating infections occur, the prognosis is better when patellectomy has been performed; (*e*) excision of the patella does not impair the efficiency of the knee.

7. Regeneration in kind may occur following excision of the patella to protect the exposed condyles.—*Mary Frances Vastine.*

Cave, Edwin F., Rowe, Carter R., and Yee, Lester B. K. Chondromalacia of the patella. *Surg., Gynec. & Obst.*, Oct., 1945, *81*, 446–450.

Chondromalacia of the patella is a pathological entity and may be a cause of internal derangement of the knee. Of 124 consecutive knee arthrotomies performed at the 105th General Hospital for internal derangement, there were 9 cases (7 per cent) in which the cause of the derangement was found to be degeneration of the articular cartilage of the patella. This relative frequency of occurrence suggests that the condition in the adolescent or the young adult is not uncommon and is frequently overlooked because: (1) while the history is suggestive of internal derangement of the knee, the physical examination is inconsistent, largely because of the difficulty in examining the articular surface of the patella; (2) the roentgenogram of the patella is negative; (3) at operation, adequate exploration of the patella is frequently not carried out.

Pathological Changes

1. The typical appearance of well advanced chondromalacia of the patella resembles shredded crab meat. The articular surface of the patella presents a softened area with multiple striated fractures in the hyaline cartilage with particles of cartilage attached at the margin of the area.

2. Not uncommonly, small cartilaginous bodies are detached from the degenerated area and are found free in the knee joint. As time goes on these loose bodies may be partially or completely calcified.

3. In the early stages, changes in the joint may remain confined entirely to the patella but after months or years, thickening and congestion of the synovia may result with eburnation of the margins of the femoral and tibial condyles and partial destruction of the articular cartilage. The latter changes are most marked in the femoral condyles where they are in contact with the gliding patella.

Diagnosis

1. The diagnosis of chondromalacia of the patella should not be confused with injury to the semilunar cartilages although both conditions may exist in the same joint. One should exercise great care in palpating the articular surface of the patella by displacing it from side to side during the examination. Local tenderness under the patella may be the only positive finding, and even this may not be present if the pathological changes are confined to the center of the articular surface. It may be possible to

produce pain by pressure or percussion over the patella with the knee acutely flexed.

2. *Mechanism of injury.* In this series of 11 cases, symptoms were precipitated by an indirect twist of the knee in 5, a direct blow on the patella in 5, and in 1 there was no definite history of injury.

3. *Symptoms.* Pain was present in all cases. Seven had recurrent swelling, 7 had infrequent catching or a sensation of weakness or "giving away" of the knee and only 2 had a history of actual locking of the joint. In both of these cases loose bodies were found at operation.

4. *Physical examination.* On this examination, 7 patients presented increased synovial fluid, 6 had atrophy of the thigh. Given a patient with a history of intermittent pain over the anterior aspect of the knee associated with a momentary sensation of catching, followed perhaps by slight stiffness and moderate swelling of the joint, with atrophy of the thigh and tenderness along the mesial border and articular surface of the patella, one should strongly consider the diagnosis of chondromalacia of the patella.

5. *Roentgenographic findings.* In all 11 cases roentgenograms of the patella were negative. In 3, however, there was evidence of osseous loose bodies in the joint which presumably took origin from the inferior surface of the patella. In 1 there was moderate calcification of the tibial collateral ligament.

Treatment

When the symptoms justify operation, a parapatellar incision should be used in order to allow adequate exploration of the patella. If the condition remains unrecognized and untreated the knee will develop progressive changes resulting in what may be a severe degree of degenerative arthritis. The form of treatment should be determined by the severity of the symptoms and by the extent of the changes in the patella found at the time of operation.—*Mary Frances Vastine.*

NACHLAS, I. WILLIAM, and OLPP, JOHN L. Paraarticular calcification (Pellegrini-Stieda) in affections of the knee. *Surg., Gynec. & Obst.*, Aug., 1945, *81*, 206–212.

Twenty young men with this condition were thoroughly studied by the authors.

Roentgenographic Examination. The calcified masses in Pellegrini-Stieda disease are seen to be situated in the soft tissues on the medial aspect of the knee, directly overlying the adductor tubercle. A radiolucent area exists between the condyle and the calcareous mass. This translucent area is usually less than a millimeter in width. The shadow may appear as a straight line. When it is more than a centimeter in length it appears to have the shape of a curved line and is more or less parallel to the curve of the tubercle. The tissue in which the lime salts are deposited is not part of the bone but overlaps the tubercle fairly intimately so that it is convex.

Operative and Autopsy findings. The Pellegrini-Stieda calcification is formed directly under a membrane that overlies the lower femoral condyle on its medial aspect. Lateral to it is the periosteum of the adductor tubercle. When the calcific mass elongates, it extends in a proximal direction so that it lies between the tendon of the adductor magnus and the overlying membrane. The condition is not due to a fragment of bone from the femur, a tear of the periosteum, or a calcified bursa.

Symptoms and Physical Findings. When asked to localize the pain in the knee these patients pointed to the area just below the patella. None of them indicated the region of the adductor tubercle.

Many of the men had some discomfort in the knee at the extremes of extension and flexion. Some of them had an ache that was present particularly on weather changes and when they first arose from a sitting position. Others complained that prolonged walking made the knee hurt.

Summary. The name "Pellegrini-Stieda disease" is misleading in that it implies that the one finding of the calcification over the adductor tubercle is a disease complex. The stress that is given this particular manifestation tends to detract from what is usually the significant part of the patient's disease, namely the arthritis of the knee joint. Furthermore, because of the visualization of this anomaly, there has been a tendency to associate the symptoms of the affected knee with it. This belief has led to surgical intervention for the removal of the mass. In the authors' opinion such an approach to the problem is inaccurate in that it stresses a relatively innocuous manifestation and tends to overlook the major disabling factor.

A process of attrition takes place on the inner aspect of the gliding membrane over the critical

point, namely the adductor tubercle, where friction can most easily be obtained. This process may have its origin in a series of small traumas that do not attract much attention. The process of attrition will be speeded by the swelling of the tissues that is induced by either polyarticular arthritis or local traumatic arthritis of long standing. As a result of the degenerative process affecting the undersurface of the membrane, the tensile strength is reduced so that a relatively moderate injury may cause an incomplete tear. This rupture is associated with a laceration of small blood vessels permitting a moderate amount of bleeding to take place. The small hemorrhage is trapped locally and is molded in its position by the outer coating of the membrane. The encapsulated hematoma thus formed undergoes degenerative changes which, under the proper physical and chemical conditions, lead to the calcification.

It is significant that in all of these patients there was evidence of arthritis in the adjacent knee joint. Examination revealed thickening of the capsule, occasionally there was fluid increase, and on rare occasions there was limitation of action. All roentgenograms showed the bony lippings that are considered manifestations of arthritis. Since these changes are not produced immediately after an injury it must be deduced that joint inflammation had been present for some time prior to the precipitating injury. Indeed, one was able to obtain from the patients the history that the knee joints had been troublesome for months or even years before the examination that revealed the calcific masses. The pains in the knees were chronic, some being attributed to a weak joint while others were considered rheumatic. Since chronic inflammatory involvement of the knee was always co-existent it deserves consideration as an etiologic factor.—*Mary Frances Vastine.*

Ver Brugghen, A. Massive extrusions of the lumbar intervertebral discs. *Surg., Gynec. & Obst.*, Sept., 1945, *81*, 269–277.

A great deal has been written on the subject of the ruptured intervertebral disc, but it appears that insufficient stress has been laid on the serious complications which may occur when this condition is inadequately treated. The extrusion of a large part of a disc may so compress the cauda equina as to cause weakness of one leg with disturbance of urinary function or even a transverse cauda equina lesion with paraplegia and incontinence of the bladder and rectum.

Nine such patients were seen by the author. Eight of these were operated upon and one died before anything could be done. These 10 cases represent the serious complication seen in perhaps 300 consecutive cases of typical herniated nucleus pulposus (associated with sciatica and without bladder symptoms or incapacitating weakness).

Age and Sex Incidence and History. Half of this series of patients included men and half women. The ages varied between thirty-five and forty-five years. The history was of the "cogwheel" variety in most instances in that the picture developed in successive acute short episodes. There was a history of backache, usually with sciatica, in all but one case. A history of trauma was obtained in 6 of 9 patients. Most of the patients had pain on coughing or sneezing. There was serious bladder involvement in all but one.

Signs. The signs were those of pressure on the cauda equina, viz., weakness and hypesthesia (which was rarely bilaterally symmetrical). The Lasègue sign was positive in all cases and deep reflexes of the ankle were affected in every instance. Six patients had a tender spine on deep pressure.

Roentgenologic Findings. Five of 9 patients showed a narrowed intervertebral space at the site of the herniation of the involved disc. The location of the extrusion was at the lumbosacral joint in 3 cases, between the fourth and fifth lumbar vertebrae in 3 cases, and between the third and fourth lumbar vertebrae in 2 cases.

Differential Diagnosis. Metastatic malignancy. In both ruptured disc and metastatic involvement there may be a short history of pain in the back with pain down the legs followed closely by weakness and sphincter disturbance. The histories are dissimilar in that the ruptured disc comes on in one or two, or a series of incidents whereas the metastatic lesion usually presents a steadily progressive history.

Pott's disease of the spine may cause cauda equina signs and symptoms but the history is usually shorter.

Spinal cord tumor must receive consideration. The Lasègue sign is uncommon in cord tumors. (In a footnote, the author states that Lasègue's sign is used to mean passive, straight leg raising with the patient in the horizontal position. He notes that this is not the sign de-

scribed by Lasègue which consisted in flexing the thigh on the abdomen and extending the leg on the thigh. Except in disease of the hip, the conclusions in the two maneuvers are similar.) —*Mary Frances Vastine.*

BLOOD AND LYMPH SYSTEM

CLARKE, HAROLD M. Primary carcinoma of the extremity. *Surg., Gynec., & Obst.*, Dec., 1944, *79*, 669–672.

Of the 214 cases studied (4 per cent of an estimated 5,000 cases of skin cancer recorded at the Barnard Free Skin and Cancer Hospital from 1925 to 1940), in only 15 per cent was the lower extremity affected as compared with 85 per cent for the upper extremity. Of the 182 cases of upper extremity carcinoma, 132 occurred in males and 50 in females, whereas in the lower extremity there were 23 cases in males and 9 in females. The lesion of the upper extremity is definitely one of advanced age as the maximum incidence occurred in the group of patients seventy years of age or older. The incidence of lower extremity carcinoma is greatest in the group fifty to sixty years of age.

On the upper extremity, the preponderance of lesions occurred on the dorsum of the hand. There were 150 lesions of the hand, 24 of the arm, and 9 of the fingers. In this clinic, serving largely a rural population, a great many cancers have occurred as a result of exposure to solar radiation. Of the 182 patients with upper extremity lesions, 72 gave a history of being farmers or had been engaged in outdoor occupations; 59 exhibited or gave a history of pre-existing keratoses and 50 had already either experienced or subsequently developed another cutaneous cancer.

The malignant lesion of the extremity is usually a squamous cell crcinoma. There were 163 such lesions of the upper extremity as compared with 18 basal cell lesions.

Treatment. The treatment of all but 7 of the local lesions was surgical and consisted of either local excision or amputation, depending on the extent of the involvement. In general, the decision to perform an amputation rested upon the amount of fixation of the lesion.

The proper handling of the lymphatic drainage areas of the extremities is the most perplexing problem offered in connection with the treatment of carcinoma of the extremity. In general, it may be said that in this series lymph node dissections were not done unless there were clinically involved nodes.

Prognosis. The prognosis of the disease, as shown in this series, has been good in regard to the upper extremity, but less favorable in regard to the lower extremity. In the followed group of 136 cases in which the upper extremity was involved, the survival rate was 93 per cent. In the 22 followed cases of lower extremity involvement, the survival rate was 70 per cent.—*Mary Frances Vastine.*

VORIS, HAROLD. Subdural hematoma. Editorial. *Surg., Gynec. & Obst.*, July, 1945, *81*, 99–101.

The hemorrhage in this condition is not necessarily traumatic. Carcinoma or sarcoma involving the dura, neoplasm or vascular malformation may occasionally be the predisposing factor.

It has been assumed that the initial lesion is the escape of blood into the subdural space. As the result of a fall or a blow to the head the brain is displaced in the cranial cavity. This may cause tearing of a vein crossing the subdural space. Usually it is a superior cerebral vein that is torn but inferior veins to the sphenoparietal or transverse sinuses may be torn. The venous pressure in the cerebral veins is low and the bleeding usually soon stops spontaneously. As liquefaction of the blood in the subdural space begins the resultant fluid is high in protein content. Since it is separated from the subarachnoid space by only the arachnoid and since this is impervious to the large protein molecules in the liquefying blood, an osmotic imbalance must exist between the cerebrospinal fluid and the liquefying blood with resultant passage of fluid into the subdural space. As time goes on and the hemoglobin molecules begin to break down, there is a great secondary increase in molecular concentration following the breaking down of the large protein molecules. Consequently there is a late secondary tendency to increase in size of the clot. The dura reacts early to the presence of the blood in the subdural space, and a neomembrane or capsule forms first on the dural side. Later a more gradual reaction of the arachnoid produces a neomembrane on the arachnoid side of the clot.

Most subdural hematomas are on the lateral aspect of the hemisphere; some are inferior and anterior, others posterior and inferior. They are progressive expanding lesions and must be treated surgically. A burr hole in the parietal region above the ear will disclose practically every clot.

History. In 75 consecutive patients who were operated on a history of trauma was present in all but three. At times the trauma is so slight that it is overlooked by the patient, particularly if a considerable time interval intervenes between the trauma and the appearance of the symptoms. This interval may be brief or it may range up to several months. Time intervals of a year or more raise the question of a second later, but forgotten, injury.

Signs and Symptoms.

1. Mental disturbance was noted in over 90 per cent of the cases. The picture is usually one of progressive confusion finally advancing to stupor or coma. Often the condition fluctuates markedly. One day the patient is grossly confused or stuporous, the next relatively alert.

2. Neurologic abnormalities in the form of hemiparesis or paralysis, abnormal reflexes or some degree of aphasia are very frequent. These findings often do not appear until late in the development of the condition. Headache, dizziness, or nausea and vomiting are common.

3. The spinal fluid is xanthochromic in two-thirds of the cases. Clear spinal fluid does not rule out the presence of a hematoma or even make it improbable but xanthochromic fluid usually indicates the diagnosis. However, subarachnoid hemorrhage is often present in brain injury and, if the antecedent trauma precedes the spinal puncture by less than two weeks, the xanthochromia has to be disregarded.

4. Papilledema is found in one-half of the cases. Convulsions, often jacksonian, sometimes generalized, are seen nearly as often as in tumors near the central sulcus. Occasional bradycardia with a pulse rate below 60 is present in many cases. It is seldom persistent, only recurrent.—*Mary Frances Vastine.*

Lesh, Ruth Ellis. Mesenteric thrombosis complicated by dermoid cyst. *Am. J. Obst. & Gynec.*, Oct., 1945, *50*, 422–426.

The author gives a brief review of the literature pertaining to mesenteric thrombosis:

Age incidence—The average age is forty-three to forty-five years and males predominate by about 40 per cent.

Mesenteric occlusion may be arterial, venous, or a combination of both. In arterial occlusion either embolism or thrombosis may precede, while in venous occlusion thrombosis has always occurred. The resulting infarction usually leads to necrosis of the adjacent intestinal wall with resulting peritonitis and gangrene. The affected bowel then becomes dark red, with swollen mucosa and a lumen filled with hemorrhagic material.

The intestine involved may be anywhere from a few inches to the entire small bowel. Brown mentions the average length of affected intestine in 22 patients who recovered as 38.1 inches, and as 102.4 inches in 39 who died.

Causative lesions of venous and arterial occlusions of the mesenteric vessels are many. Among them are portal obstruction, cardiac conditions, septic abdominal lesions, intimal lesions, peripheral sepsis, degenerative diseases such as arteriosclerosis, and mechanical damage arising from hernia, surgery, and obstruction.

In its clinical features, mesenteric thrombosis is characterized primarily by pain, which may be dull and cramping or sudden and severe. It is usually centrally located, incessant, and not accompanied by muscular spasm or extreme tenderness before gangrene and peritonitis develop.

Localization of pain does not occur early and therefore rarely gives a definite clue for early diagnosis. When the pain is localized it is more often in the lower right quadrant. Klemp believes that thrombosis of the inferior mesenteric vein gives rise to pain predominantly on the left side, while thrombosis of the superior mesenteric vein produces pain mainly on the right side.

Three points upon which authorities agree as distinctive features of the pain of mesenteric occlusion are: (1) that it is more severe than the clinical findings warrant; (2) that it is not alleviated by morphine; and (3) that there is much less tenderness than would be looked for with such intense pain.

Vomiting is more common in arterial than in venous occlusion. Constipation or diarrhea accompanied by bloody stools may be coincident, the former being more common.

Distention is probably present in less than half the cases and is usually moderate, being more frequent in venous than in arterial thrombosis.

The leukocyte count and pulse rate furnish valuable clues to mesenteric occlusion because they are both almost uniformly high out of proportion to the temperature, which, like the blood pressure, is usually low.

The differential diagnosis includes ruptured peptic ulcer, various blood dyscrasias, and

pelvic conditions such as ectopic pregnancy and torsion of pedicle of ovarian cysts.

The treatment of mesenteric occlusion is, of necessity, surgical.—*Mary Frances Vastine.*

GENERAL

WILSON, DAVID A. Tumors of the subcutaneous tissue and fascia, with special reference to fibrosarcoma—clinical study. *Surg., Gynec. & Obst.*, May, 1945, *80*, 500–508.

This study is based on a review of tumors of the subcutaneous tissue and fascia seen in the Surgical Out Patient Department and in the Surgical Pathology Laboratory of Duke Hospital from 1931 to 1941. There were 871 cases out of 80,000 surgical specimens studied.

The ratio of benign to malignant tumors was 8 to 1. The diagnosis is frequently established only after the tumor attains considerable size or the overlying skin becomes ulcerated. Every relatively cellular fibroma, no matter how casual its clinical or histological appearance, should be considered malignant until the subsequent behavior is established.

Incidence. The relative incidence of the various connective tissue tumors in this series was as follows: Of 871 cases, 111 were fibrosarcomas, 142 fibromas, 358 lipomas, 142 hemangiomas, 17 lymphagiomas, 39 angioendotheliomas, 31 neurofibromas, and 31 neurilemmomas.

Sex. Fibrosarcomas occurred 68 times in the male and 43 times in the female, a ratio of 1.6 to 1. This is in distinct contrast with the occurrence of benign tumors, which occurred more often in females or in equal sex distribution.

Race. Fibrosarcomas occurred 39 times in negro and 82 times in white patients. The incidence was slightly higher in the white race than in the colored race when compared to the ratio of white patients (62 per cent) and colored patients (38 per cent) seen in the clinic annually.

Age. In this series, an almost equal distribution of fibrosarcomas occurred between the ages of ten and seventy, the average being 36.4. The age incidence of fibrosarcomas closely paralleled the age incidence of benign fibromas.

Relation of Trauma. There is evidence to support the view that trauma may be a factor in the formation and acceleration of many benign subcutaneous tissue tumors. The relationship of trauma to the formation or acceleration of fibrosarcoma is doubtful.

Gross Characteristics. Primary fibrosarcoma arises most frequently in the subcutaneous tissue and fascia and less frequently in the tendons, dermis, and muscle. It appears to have a predilection for the upper thigh and shoulder region, occurring to a lesser extent around the knee and elbow. Fibromas occur more frequently around the head, hands, feet and legs. Lipomas show a higher incidence on the body.

Fibrosarcomas are usually single. They grow expansively and in a major portion of cases appear to be encapsulated. Invasion of the surrounding tissue is the rule.

In this series, 90 per cent of fibrosarcomas measured over 5 cm. in diameter when the patients first presented themselves for examination.

Microscopic Characteristics. Fibrosarcomas grade from very cellular anaplastic types to fibrogenic tumors difficult to differentiate from fibromas. The relative proportion of cells to fibrillar intercellular substance is undoubtedly the best single criterion for estimating the growth potentialities of fibrosarcomas.

Metastases. Metastases in fibrosarcoma usually occur late by way of the blood stream. The most frequent site of spread is to the lungs. The regional lymph nodes may be involved. Occasionally metastases are found in the subcutaneous tissues before pulmonary metastases become evident.

Differential Diagnosis. Fibrosarcomas may be differentiated from fibromas by their size, by their more rapid expansile type of growth; by their characteristic location and by their tendency to invade and destroy skin and bone.

Treatment. Of utmost importance is the early recognition of the malignant nature of the tumors and wide surgical excision. Roentgen therapy in the treatment of fibrosarcoma is without appreciable value.—*Mary Frances Vastine.*

ROENTGEN AND RADIUM THERAPY

SNITMAN, MAURICE F. Individualization in the management of carcinoma of the maxillary sinus. *Ann. Otol., Rhin. & Laryng.*, March, 1945, *54*, 125–135.

One of the main objectives of this paper is to call attention to a plan of management suggested by Dr. Danely P. Slaughter, Director of the Tumor Clinic at the University of Illinois College of Medicine. This plan consists of the application of radium within the cavity as a supplementary measure to external protracted irradiation, after exposure of the antrum by conservative surgical intervention. The results of this technique have been highly satisfactory

and have obviated the necessity of radical operation.

A line extending from the inner angle of the eye to the angle of the jaw divides the antrum into a suprastructure and an infrastructure. Lesions above this Ohngren malignancy plane, situated medially, are frequently associated with nasal polyps and encroach on the meninges more readily and tend to early lymphatic invasion. In the lateral part of the suprastructure, carcinoma has, at its onset, the most dormant development. It then rapidly invades the malar bone producing the characteristic tumefaction of the external angle of the floor of the orbit. Malignancies in the infrastructure of the antrum produce earlier symptoms referred to the teeth.

Treatment. In the consideration of a therapeutic regimen, it is essential to consider the site of the primary lesion and the presence or absence of lymphatic extension.

Lesions involving the roof of the sinus present a most unfavorable prognosis and it is unfortunate that this area is the affected site in most cases.

Lesions in the floor of the sinus with involvement of the hard palate and alveolar ridge are amenable to intra-oral surgical removal, followed by local radium application.

Treatment of the primary lesion is not affected by the presence of regional metastases. If metastases are present, a new therapeutic problem presents itself. Carcinoma of the head and neck, if untreated, produces metastases to the cervical lymph nodes. The latter serve as protective barriers to the spread of cancer.

Patients with cancer of the head and neck present one of three conditions in the cervical region: (1) no palpable metastatic nodes; (2) operable nodes; (3) inoperable nodes.

In managing the lymph nodes, no prophylactic dissection or irradiation is performed. If the nodes are operable and the control of the primary lesion is assured, neck dissection is performed. If the nodes are inoperable, the patient is submitted to external irradiation supplemented with radon seed implantation.—*Mary Frances Vastine.*

Smith, G. V., and Dresser, Richard. Carcinoma of the uterine cervix. *Am. J. Obst. & Gynec.*, July, 1945, *50*, 1–10.

Eleven hundred and eleven consecutive, previously untreated, pathologically confirmable cases of cancer of the uterine cervix are reviewed. The percentages of patients who were alive five years after first treatment are as follows: 1902 to 1913, 12; 1914 to 1918, 19; 1919 to 1923, 28; 1929 to 1933, 38; 1934 to 1938, 43. The percentage of patients alive at ten years in the same groups to 1934 are: 7, 11, 12, 21, and 29. The improved results are due for the most part to the use of radium and roentgen rays. Seventy-one per cent of the patients alive five years after treatment were alive at ten years, and 75 per cent of those alive at ten years were alive at fifteen years.

Deaths and Complications from Irradiation

1. Overtreatment.
   a. The authors now consider anything over 5,400 mg-hr. given in one application and anything over 6,000 mg-hr. given in two applications an overdose of radium. As a rule, no amount of radium is left in over thirty hours.
   b. Despite the restrictions outlined in a, excessive damage will ensue in some patients even if roentgen rays are not administered. The maximum relatively safe amount of 200 kv. roentgen radiation, when combined with the above dose of radium, is 6,000 r, measured with scattering, about one-third of which is delivered to the center of the pelvis.
   c. It appears that the maximum relatively safe dose of 1,000 kv. roentgen radiation is about 7,200 r, measured with scattering, about one-half of which is delivered to the center of the pelvis.
   d. The maximum relatively safe daily dose of roentgen radiation is 400 r.
   e. It is felt that it is better to give all radiation within six weeks and to administer roentgen therapy first except in cases with hemorrhage, which is more quickly controlled by radium.
   f. If the above amounts of radium and roentgen rays do not destroy a given cancer, it is likely that more will be ineffective.
   g. Of course, some patients can tolerate more irradiation than others and some cancers respond better than others to the same dosage.
2. Retreatment.
   a. In general, retreatment is only occasionally really beneficial and, at that, usually only for local vaginal recurrences. Hence, it is safer to withhold additional therapy

unless the evidence for recurrence is convincing.

3. Delaying Operative Interference in the Hope that Complications Would Improve Spontaneously.
   a. In retrospect it seems that nearly all the deaths which were attributed to radiation could have been prevented by timely colostomy and/or diversion of the ureters.
   b. Since most of the deaths and potentially lethal complications were due to ureteral damage, the development of irradiation reactions is a signal for repeated intravenous pyelography and preparedness for surgery as long as the patient lives.
   c. Ureteral stenosis can occur quickly and at any time up to fifteen years after treatment.

Treatment of Irradiation Complications

1. Acute post-irradiation pelvic inflammations have been the cause of nearly all the immediate and early deaths. Fortunately these have been rare. Because perforation of the lower bowel is very likely involved, the authors think pelvic drainage and colostomy may be salutary, along with the modern antibacterial drugs.
2. Irradiation proctitis: The tenesmus and pain, though usually of less than two months' duration are difficult to relieve. One grain opium suppositories seem to be as effective as anything. For the usual not alarming bleeding, iron, liver and high vitamin diet are prescribed. For the rare profuse and prolonged rectal bleeding, repeated transfusions of whole blood are given.
3. Irradiation cystitis: The authors are trying frequent instillations of purified cod-liver oil for this complication.—*Mary Frances Vastine.*

McLENNAN, CHARLES E. Results of various types of treatment in adenocarcinoma of the endometrium. *Am. J. Obst. & Gynec.*, Sept., 1945, *50*, 254–262.

The results of treatment in the University of Minnesota Hospital of 225 patients with adenocarcinoma of the uterine corpus have been reviewed. The absolute five year cure rate has been 45 per cent.

Poor results are shown to follow routine use of radium and roentgen irradiation alone. Certain patients so treated may be saved many years later by hysterectomy for recurrent or, perhaps, persistent carcinoma. Good results have followed the use of total hysterectomy, with or without preoperative or postoperative irradiation.

Discussion. Hysterectomy is conceded to be the important part of the therapy for carcinoma of the uterine body. In recent years the only objection to this concept has been raised by Heyman at the Radiumhemmet in Stockholm where specialized techniques for the application of intracavitary radium are employed in the primary therapy.

The question to which an answer is being sought is whether the preoperative addition of radium and/or roentgen therapy will materially increase the survival rates in operable cases.

The immediate undesirable effects of giving full-tolerance doses of roentgen radiation to patients with carcinoma of the uterine corpus suggest that persistence with this part of the therapy will add little or nothing to the ultimate cure rates. In this small series the primary mortality attributable to irradiation was excessive (8 per cent), and the morbidity was likewise great. One gets the impression that the acute complications of radiation therapy are comparatively great in elderly women of poor general physical status, many of whom are obese.—*Mary Frances Vastine.*

PARKS, THOMAS J. Carcinoma of the ovary treated preoperatively with deep x-ray. *Am. J. Obst. & Gynec.*, May, 1945, *49*, 676–685.

The treatment of cancer of the ovary is notoriously unsatisfactory. Probably less than 10 per cent of the solid infiltrating type and 40 per cent of the papillary adenocarcinomas survive five years. Without exception, all who have studied the problem agree that in the treatment of carcinoma of the ovary, excision is the procedure of choice. Irradiation for these cases is not so well established. Some support its use as an adjunct to operation, while others admit no benefit from irradiation and assert that it can cause harm.

The prevailing practice in ovarian neoplasms is to operate, to remove as much of the mass as possible even though large residual carcinomatous masses are left in situ, and then to employ roentgen irradiation.

Report of Cases. Three cases are presented in which the history and physical examination were comparable. In each instance, because of

the size of the growth, density of adhesions, extent of infiltration and the apparently hopeless outlook, the operator did not remove the pelvic organs. A biopsy was taken and deep roentgen therapy was given which obviously caused the masses to become smaller and more movable and the implants on the peritoneum to shrink. Several months after the first operation, a second operation was performed in each case and at this time the neoplastic tissue and affected organs were removed. Two of the patients are still alive and apparently well after twelve and eight years. The third patient died of a carcinoma, thought to be unrelated, five years after the original operation.

Conclusions. Deep roentgen therapy in the 3 cases reported caused the papillary excresences and transplants to shrink and the entire mass to diminish in size, thus making operation easier.

This study indicates that it is unwise to persist in doing a difficult primary operation. In some cases of massive infiltration, it may be safer to take a biopsy, close the abdomen, give deep roentgen therapy and perform a second operation at a later time.—*Mary Frances Vastine.*

Bethell, Frank H. Leukemia: the relative incidence of its various forms and their response to radiation therapy. *Ann. Int. Med.*, May, 1943, *18*, 757–771.

The series in this report is composed of 495 cases examined at the Simpson Memorial Institute between July 1, 1927, and December 31, 1941, a period of 14½ years. For the purpose of this and subsequent studies the records of all patients were analyzed and data supplied by the history, physical examination and laboratory procedures including biopsy and necropsy, were tabulated.

All of the cases observed were classified according to the three parent cell types: the lymphoblast, the myeloblast, and the undifferentiated or but slightly differentiated reticulum cell (histioblast or histiocyte).

The relative and absolute increase in the acute forms of leukemia observed in recent years is believed to indicate a greater incidence of such diseases.

The indications for radiation therapy in myelocytic and myelomonocytic leukemias are symptoms of anemia, of increased metabolism, of pressure from splenomegaly, and of pain in or referred from the splenic area. In the absence of disabling symptoms therapy is deferred. The level of the leukocyte count at the time of institution of treatment bears little relation to the indication for its use, but the rapidity of decline of the white blood cell count is a useful guide to its continued employment. It is pointed out that the value of radiation therapy is limited to patients with the chronic forms of leukemia. Best results are obtained in the myelocytic and lymphocytic varieties, but the degree of improvement, the duration of the remission, and the time which will elapse before the occurrence of a refractory state cannot be foreseen in any individual case.—*James J. McCort.*

Uhlmann, Erich M. Alpha rays in the treatment of wounds. *Surg., Gynec. & Obst.*, Oct., 1944, *79*, 412–418.

As long as twenty years ago, Strasburger introduced radon in a liquid vehicle into therapy. For the past fifteen years, radon dissolved in fat substances has been successfully used for the treatment of skin changes and ulcerations which resulted from overdosage of roentgen rays and radium. These ulcerations are characterized by the pain associated with their appearance, their resistance to therapy, and by their eventual development into cancer with all its consequences.

It may seem odd and inconsistent to attempt the cure of injuries produced by roentgen rays or radium with the same agent, but it must be emphasized that the recommended therapeutic factor for these skin changes consists of alpha rays which are corpuscular elements and physically different from gamma rays of radium or roentgen rays. In the dosage employed for these treatments, alpha rays have been proved to be absolutely harmless in many thousands of applications.

Radon incorporated in neutral fat is a source of alpha rays and has the appearance of an ointment. It can be handled like other salves but consideration must be given to the fact that it is not a stable product. Radon disintegrates and loses its strength by continuously giving up part of its charge. This loss has been exactly determined and amounts to about 16 per cent in every twenty-four hours. An ointment with a known amount of radon will lose half of its concentration in about four days. The actual

amount employed for therapeutic purposes here described is exceedingly small. Radon in this form is measured in electrostatic units (e.s.u.), 1 electrostatic unit being equal to 0.000364 millicurie-destroyed. Ten cubic centimeters of radon ointment containing a total of 1,000 electrostatic units (100 e.s.u. per cc.) can be applied continuously for more than 100 hours before an erythematous reaction of the skin will be produced. Radon is a gas so tends to escape. Therefore, in treatment, the dressing over the ointment must be made as airtight as possible. Repetition of the treatment is usually done at intervals of three to seven days. The treatment time depends upon the nature of the lesion, and the number of treatments required is influenced by its size.

The use of radon ointment was originally limited by the author to the treatment of wounds which had proved to be resistant to all other forms of therapy and in cases in which wide excision of tissue with subsequent grafting of skin or even amputation of limbs was contemplated. Most of these lesions were sequelae of overdosage of roentgen rays or radium. The results obtained were extremely gratifying. Consequently, when radon in this form became more easily obtainable, other skin conditions with similar resistance to ordinary therapy were subjected to these treatments. Periodic histological studies of such lesions showed that new capillaries were formed under radon ointment therapy. Therefore, these treatments were instituted in ulcerations of the skin where the local blood supply was incompetent. In this manner patients suffering from Buerger's disease, varicose ulcers, necrotic lesions including diabetic gangrene, resistant burns resulting from heat or electric current, and draining fistulas were successfully treated.

During the past year the influence of alpha rays on experimental surgical wounds in rabbits' skin was studied and valuable information on the subject of wound healing and scar formation obtained. These wounds did not always correspond to changes in human tissue, but in the response to radon ointment therapy a surprising similarity in rabbits' and human skin was observed. Uniformly, the healing process of extensive wounds was accelerated by the application of radon ointment.

Clinical results obtained in surgical wounds selected on the basis of experience gained in the experimental work, confirmed the value of radon ointment applications. The rate of wound healing was greatly hastened and surprisingly soft scars were produced without impairment of function.—*Mary Frances Vastine.*

PRUDENTE, ANTONIO. Postoperative prophylaxis of recurrent mammary cancer with testosterone propionate. *Surg., Gynec. & Obst.*, June, 1945, *80*, 575–592.

Endocrine Dysplasias of the Breast. Functional mastopathy results from a disturbance of the hormoral balance. The causes may include: (1) excessive blood estrogen, (2) excessive luteal hormone in the blood, (3) ovarian insufficiency, (4) of thyroid origin, (5) of pituitary origin and (6) of other origin. The author feels that over 50 per cent of the cases of mastopathy are caused by hyperestrinism. Thus, the failure of estrogen therapy in many cases of mastopathy is understandable.

The use of the antagonistic hormone, testosterone, gives extraordinary results in most cases of mastopathy.

Mastopathy and Cancer. Cole and Rossiter believe that 20 to 30 per cent of cases of mastopathy are of the "precancerous hyperplasia" type.

Warren studied 1,044 cases and states that cancer occurs 4.5 times more often in individuals with mastopathy than in healthy women.

The author is convinced that there is a causal relationship between certain cases of mastopathy and cancer. He believes that the occurrence of cancer is higher in cases of mastopathy because hyperestrinism exists in almost every instance in both diseases.

Observations. It seems probable that the effect of testosterone in mammary cancer is mediated through the pituitary gland. (Lacassagne and others observed atrophy of the anterior pituitary in rats after testosterone administration.)

In cases in which neoplastic elements remain after operation, the administration of testosterone propionate results in the formation of dense fibrous tissue.

There is evidence of an antagonism between estrogens and testosterone.

Conclusions. The relationship between hormonal mastopathia and mammary cancer, the influence of estrogens in the occurrence of this tumor, and the biologic antagonism between androgenic and estrogenic hormones justify the use of testosterone propionate in the prophylaxis of mammary cancer after operation.

The results obtained from this treatment as gauged by the survivals after three, four, and five years are about 100 per cent better than those observed after operation only. This fact indicates that testosterone propionate exercises a protective or prophylactic action against recurrences of surgically treated mammary cancer.

Very high doses of testosterone propionate, up to 175 mg. per week, should be used after operations for mammary cancer, and this treatment should be continued through many years. The principal criterion for the selection of the dosage is the grade of histologic malignancy of the tumor.

The side effects following this therapy are of secondary importance. They consist of signs of virilization which occur almost exclusively in young women—in about 50 per cent of cases, and of disturbances of menstruation occurring in almost all patients.

In patients between forty and fifty years of age who are still menstruating, early cessation of the menses is often observed.—*Mary Frances Vastine.*

Treves, Norman, Abels, Jules C., Woodard, Helen Q., and Farrow, Joseph H. The effects of orchiectomy on primary and metastatic carcinoma of the breast. *Surg., Gynec. & Obst.*, Dec., 1944, *79*, 589–605.

This is a study of 6 males with carcinoma of the breast on whom orchiectomy was performed. The conclusions reached are as follows:

1. The clinical results of this study may be summarized by stating that bilateral orchiectomy may have been responsible for the temporary regression of the primary lesion in 2 instances. It has possibly prevented recurrence in the primary operable patient treated only by local excision. The procedure has caused regression and repair in the secondary deposits in bone as well as in lung. The clinical regressions in this study compared with those reported for inoperable and metastatic carcinoma of the prostate have had in some cases the same dramatic cessation of pain caused by bone involvement. The clinical improvement has been as satisfactory and as long.

2. In the histological examination of the testes of the 6 orchiectomized patients, essentially normal gonads were found. The mammary cancer had not metastasized to the testicles. This is in striking contrast to the findings in 33 women who were castrated for therapeutic control of breast cancer. In this group 9 patients had mammary carcinoma metastatic to the ovaries.

3. The studies on 2 of the orchiectomized patients led to a conclusion that of the metabolic phases measured—(1) fluid and electrolyte balance; (2) nitrogen balance and serum protein fabrication; (3) urinary excretion of creatine; and (4) the utilization of carbohydrate—none changed in twelve to eighteen days after operation. This failure to demonstrate changes known to be induced by the administration of testosterone or alpha-estradiol, shows that these changes are not necessary to produce clinical symptomatic improvement.

4. In 1 castrated patient the same changes in the serum alkaline phosphatase were seen which were observed regularly in patients with carcinoma of the prostate metastatic to bone following orchiectomy. In 2 castrated patients there were no conspicuous changes in the calcium, phosphorus, or alkaline phosphatase of the serum, and in 1 the findings indicated continuing progress of the disease. In the patient treated with alpha-estradiol there was a rise in serum calcium similar to that found in female patients with bone metastases from carcinoma of the breast who have received estrogens. Thus, in this small series of patients, there is a marked lack of uniformity in the response to endocrine treatment of the blood changes caused by bone metastases. Further observation will be necessary before the cause of this variability can be determined.

5. The real value of the procedure (orchiectomy) will only be determined by observations on a larger group.—*Mary Frances Vastine.*

Jones, Howard W., Jr., and Neil, William, Jr. The treatment of carcinoma of the cervix during pregnancy. *Am. J. Obst. & Gynec.*, Oct., 1944, *48*, 447–463.

The material for this study includes 48 cases, exclusive of the authors', in which irradiation was used some time following conception and prior to delivery. The authors' own material consists of 8 instances of prenatal irradiation.

It is stressed that many of the reports in the literature are invalid in a discussion of treatment in carcinoma of the cervix and are also worthless from the standpoint of prognosis for the infant because microcephaly, one of the most serious deformities caused by irradiation,

is often not recognizable at birth. A period of about three years must elapse before one can be sure that this distressing condition does not exist.

The Risk of Fetal Abnormality Following Radium Irradiation for Carcinoma of the Cervix. It is difficult, due to limitation of numbers, to express the risk of microcephaly percentagewise. If only determinate cases are considered (that is, cases reported after three years following delivery) there are 3 seriously abnormal children in a total of 15 live births, or 20 per cent. If the entire group is considered, the figure would be 3 in 52, or 6 per cent.

Five months is sometimes cited as the earliest time at which irradiation should be applied. At that time the fetus is higher in the abdomen, thus affording greater distance from the source of irradiation. There is also said to be less likelihood of abortion if ovarian function be destroyed at that time. At times abortion occurs one to sixty days following the application of radium to the cervix. It was impossible in the authors' own cases to correlate in any way dosage estimated to be delivered in the region of the child's head with the occurrence of microcephaly.

Risk to Mother. It is certain that continuation of the pregnancy by no means precludes a cure as indicated by the literature and the authors' own experience. The authors' results show 7 mothers treated during 8 pregnancies. Two are dead, 1 is living at three years, and 4 mothers treated during 5 pregnancies are living and well five years following treatment.

Technique of Treatment. If it is decided to treat during pregnancy with a view to obtaining a normal living child at term, certain points may be stressed:

1. No high voltage roentgen therapy is employed because of the dangers of substantial depth dosage in the region of the developing child.

2. The use of tubes within the cervical canal is omitted so that all possible distance from the baby may be secured and also to guard against the onset of labor by dilating the cervix.

3. As an additional precaution against stimulating uterine contractions by prolonged packing against the cervix, a large amount of radium is used for a correspondingly short period of time.

Operative Treatment. Any necessary abdominal operation either before or following irradiation of cervix carcinoma should be confined to evacuation of the uterus.

Pregnancy at Term. If the baby is viable when the carcinoma is discovered, the problem of injury to the fetus by irradiation need not be considered. Under these circumstances, the child can and often must be delivered by cesarean section. The prognosis under these circumstances has been very poor. In many instances, the poor prognosis was due to the advanced stage of growth. However, the profound circulatory changes following parturition may be an unfavorable influence in arresting the disease.

Summary and Conclusions. The risk of an abnormal fetus cannot be statistically stated at the present time with certainty, but it seems probable that its upper limit is 20 per cent. If the parents and physician for any reason wish to assume this risk, it is justifiable to proceed with irradiation during pregnancy with a view to obtaining a living child at term. If the pregnancy is at the fifth month or beyond, the risk of an abnormal child is substantially less. The risk to the mother is probably not increased by this procedure as compared with interruption of the pregnancy. The mother's prognosis is intimately related to the clinical extent of the disease. The general experience has been that most cases are discovered early (League of Nations 1 or II), and the prognosis has been parallel but somewhat lower than for corresponding but uncomplicated groups.—*Mary Frances Vastine.*